OBSERVATIONS on HEADACHE as a SYMPTOM in HIGH BLOOD PRESSURE, with SPECIAL REFERENCE to DIASTOLIC HYPERTENSION.

A THESIS - By

F. H. Patel, M.B., Ch.B., (Edin.):

M.R.C.S. (Eng.); L.R.C.P. (Lond.);

D.C.H. (Lond.)

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OBSERVATIONS on HEADACHE as a SYMPTOM, in HIGH BLOOD PRESSURE with SPECIAL REFERENCE to DIASTOLIC HYPERTENSION.

Introduction.

My interest and experience in relation to headache, extends over a period of ten years in India, and has concerned itself essentially with patients, who were suffering from high blood pressure.

On observing, that some of my patients, who were suffering from headache, usually resorted to a simple expediency measure, of tying a folded handkerchief around the head, just above the ears, which seemed to have given relief in many instances, before any other therapeutic measures could be instituted, this popular practice among the Indians, has led me to enquire into the problem of headache in hypertension.

This thesis is mainly based upon the investigation of a single common symptom; that of headache, that is, pain felt in the region of the cranial vault (Pickering, 1939). Headache, as a symptom, is frequently met with in many diseases. Sometimes, it may be the only symptom of a disease which may be of prime importance, and there is no doubt, that in the popular mind, headache and high blood pressure are closely associated.

Headache is a variable symptom in patients with hypertension, while it may be present in many patients with severe degree of hypertension and less frequent in others in whom the blood pressure may be considerably raised or even persistently high. The onset in many cases is found to coincide with the aggravation of the blood pressure. The head throbs, dizziness appears, severity increases, and persists, often situated frontally or in the occiput and not infrequently over the vertex, occasionally leading to convulsions in many cases of hypertensive encephalopathy. It is, sometimes aggravated even by shaking the head and relieved by jugular compression. (Vide - compression by tying a handkerchief).

Whilst the pathology of this most frequently encountered hypertensive headache is obscure, the frequency of this symptom is the major problem in clinical practice and requires ellucidation and remedial measures to alleviate the suffering and to relieve pain - the two important objects of medical practice.

The early morning onset, described by many hypertensive patients and the relief obtained after the patient has been up and moving about for some time, and the throbbing character, in the absence of a space-occupying lesions of the brain or an increase of pressure in the cerebro-spinal fluid, suggests

that the site of origin, although in the brain, presumably is connected with the vascular supply of the brain - the cerebral arteries or arterioles.

Pickering (1933-34 (a)), has demonstrated the surest method of citing headache, is by a sudden lowering of intracranial pressure and the conclusion was drawn by him, that the pain may be due to the stretching of sensitive nerve structures, lying close to the meningeal arteries and stretching might be due to widening of the arteries.

Some observers hold the view that the pain is induced, when the vessel walls are stretched or dilated, while others seem to suggest that it occurs during periods of vaso-constriction. In other words, related to the migraine type of pain, consequently vaso-constrictor and vaso-dilator drugs have both been employed for the relief of hypertensive headache. (Keith).

Recent observations based on the brilliant work of Wolff (1943), on neurological patients under local anaesthesia, have clarified the nature and mechanism of headaches of local origin. It has been concluded that pericranial and intracranial arteries, venous sinuses and the dura itself, are sensitive to pain, and the supra-tentorial stimulation of these tissues leads to headache, mediated through the fifth cranial nerve and felt anterior to

a line joining the ears across the vertex; while similarly provoked pain of intra-tentorial origin is carried in the ninth and tenth cranial and upper three cervical nerves, which is chiefly felt posteriorly. Most headaches are vascular in origin, and the main mechanisms involved are distension, dilation; and traction applied to the intracranial arteries. The displacement of venous sinuses, local inflammation affecting pain sensitive structures,and the pressure on cranial nerves are rare causes.

While headache commonly occurs in many persons, without any important cause, it is encountered in others, with infection and intoxication. "Headache in an adult may mean much, or nothing much; in a child watch your step". (Crosby).

Wide variations, which occurs in character, in situation and severity have to be taken into account. The writer is aware that many causes of headache are not included in the series, some, because they are sufficiently frequently met with in general practice such as headache due to occular diseases like glaucoma and others due to conditions of the central nervous system and yet others due to chronic fibrositis, anaemias and the host of other causes. In all branches of medical practice, the sum total of its effects must be enormous, in pain, in limiting the power of the sufferers, in the shrinking of issues and the loss of pleasure. The

depressive and limiting effects upon those around, are also great. The distressing intensity at times fills the whole field, while at other times, it appears to call for or receive little attention.

In briefly summarizing the various factors that constitute the clinical entity the "headache", I would place the following facts before attempting a critical analysis of the subject.

The causes of headache have been classified differently by various authorities. In the British Encyclopedia of Medicine (1941) Rolleston enumerated sixty-four headings and the French's Differential Diagnosis of Symptoms (1945) mentions seventy.

In conclusion, I have become more and more convinced that headache in hypertension is vascular in origin. From the observations on my patients, after entering the medical practice, I have striven hard to explain the phenomena, based upon my own clinical experience, and study of the literatures. As I had a personal interest in the study of headache, I might have displayed over-emphasis in illustrating some of the points, for which I crave to be excused.

Vast amount of knowledge has accumulated about hypertension in general but one notices that many of the theories and the explanations found in Text-books and reviews in medical periodicals describing at length the pathology of hypertension is confusing and conflicting.

I have similarly treated my illustrative diagrams prepared carefully after considering a large collection of case-sheets and I have laid deliberate stress on the common forms of headache, but here and there I have not omitted to mention the most uncommon forms of headache, where I thought it will illuminate my thesis.

I have tried to arrange whenever possible in a natural and logical sequence of ideas and have exercised my thoughts and the study of patients without relying chiefly on recent literature or chiefly upon my own memory.

I have also tried to follow and obey the recognized rules of clinical investigations and practice and it is difficult for me to escape from the trends of following this mode of explanation. It is to be noted, that it is with the symptom of headache that the patient presents himself, and therefore, one is obliged to explain much of it on a theoretical basis.

In managing the patients, my thoughts were chiefly set in terms of function. It is with this object in mind that I placed in the fore-ground those facts, the understanding of which, is important in the management of such cases namely headache and hypertension.

Authorities differ much in the explanation of the etiology of headache and perhaps, it is the

reason for my attempt to explain the causes of headache in hypertension based on a study of a series of clinical observations.

I have tried to keep in close conformity with the recent works and thoughts on the subject. My grateful thanks are due to my teacher* who has given me guidance and who inspired me to pursue the study and from whom I have learned much and indeed my understanding.

In presenting this thesis, I have attempted to maintain a proper balance between experience, observations, opinions and statistics. Special attention has been paid to the correlation of pathological findings with the clinical manifestations of headache, resulting from various forms of hypertension.

* Professor Murray Lyon.

The causes of headache have been classified by many workers (Symond et al.) as follows:-

Types of Headache:

1. Infective.
2. Toxaemic.
3. Neuraesthenic and Psychic state.
4. Migraine.
5. Intracranial pressure.
6. Eye-strain.
7. Dental caries.
8. Diseases of Nose and Assessory Sinus.
9. Otitis-media.
10. Extra-dural abscess.
11. Intracranial abscess.
12. Meniere's disease.
13. Traumatic.
14. Fibrositis.
15. Referred pain.
16. Intracranial Neoplasm.
17. Disease of the bones and Cranium.
18. Neuritis and Neuralgia.
19. Meningitis.
20. Headache of Vascular origin.

1. Infective:

Usually described by patient as fulness or throbbing in frontal region or distributed generally over the vertex. It is continuous rather than intermittent and tends to aggravate by physical or mental effort. Diagnosis is not difficult, if associated symptoms suggest fever, malaise, taken along with systemic examination. It is to be noted, that thermometer should not be neglected, but ambulant groups of enteric fever may present predominence of headache and at times, absence of febrile symptoms.

2. Toxaemic:

In obscure cases the possibility of uraemia should be remembered. Lead poisoning and

often alleged constipation as a cause of headache, is of course immediately relieved by defaecation, therefore it must be reflex rather than toxic.

3. Neuraesthenic and Psychic State:

This is referred more as a discomfort rather than as a pain. It is a feeling of pressure or tightness around the head. May be occipital or vertical. May be continuous and associated with depression, anxiety, indecision, defective concentration, insomnia, work appears to be eminently undesirable and the use of the word "Agony" by the patient may help to make the diagnosis. (Crosby). Persistent contraction of the frontalis muscle and frowning. The patient may describe the sensation "like a nail or wedge being driven into the skull". Persistent "Neuralgic" pains, associated with hyperaesthesia of the scalp and failing to respond to analgesies, may be encountered in hysteria.

4. Migraine:

Short time attacks, separated by intervals of complete freedom. Family history, occurrence of vomiting, aura if present are distinctive.

5. Increased Intra-cranial pressure:

Generally throbbing, bursting and piercing, feeling often more severe in frontal and suboccipital region, aggravated by physical effort, stooping, straining and associated vomiting. The other

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5. Increased Intra-cranial pressure:

Generally throbbing, bursting and piercing, feeling often more severe in frontal and suboccipital region, aggravated by physical effort, stooping, straining and associated vomiting. The other

symptoms are those resembling space-occupying lesion. Fundal examination is necessary and papilloedema should be looked for, though the absence of papilloedema does not rule out the possibility of intra-cranial tumours.

It is also an essential feature of hydrocephalus and lumbar-puncture may reveal its presence.

Lowered intracranial pressure may produce headache, for example after lumbar-puncture. This is intensified by sitting or standing but relieved by lying flat, or with feet raised above the level of the head. Low pressure headache occurs in anaemias and also induced in others by taking saline purgatives.

This is physiological, due to disturbance of the normal relationship between the pressure inside an artery and that outside, as indicated by tipping the legs up, the headache would disappear. Another instance is, if the pressure was raised by compression of the jugular veins, the headache would be aggravated because of the low pressure outside the veins and compressing the jugulars merely increased the discrepancy.

6. Eye-strain:

Usually noticed early morning and follows prolonged visual attention. Acute glucoma should be sought for.

7. Dental Caries:

The molar tooth may be responsible. From the lower jaw the pain refers to the temple. From the upper jaw, the pain may be referred to behind the eye.

8. Diseases of Nose and Accessory Sinus:

Frontal sinusitis referred to supra-orbital region. Coryza may be present. Chronic empyema of one of the frontal sinus may occur. Infection of the ethmoidal or sphenoidal sinus may be present.

9. Otitis Media:

Pain in this condition may suggest extension of infection and involvement of the mastoid cells, but petrous bone especially its apex is affected. The pain is intermittent and often worse at night.

10. Extra-dural abscess:

In the middle fossa, it causes headache. In the posterior fossa, the pain referred mainly to suboccipital region.

11. Intracranial Abscess:

It may be difficult and indistinguishable from that produced by intracranial neoplasm, but frequently the intracranial abscess is subacute and the headache tends to be constant and of increasing

severity, so that it should be carefully distinguished from intracranial neoplasm.

12. Meniere's disease:

Usually unilateral headache, often continuous and referred to the same side as the diseased labyrinth, associated with vertigo.

13. Trauma:

Headache may be the type already described as neuraesthenic or may be sharp and shooting, circumscribed and referred to the site of injury and may even present itself long after injury. In the more severe degree of head injury, headache is apt to be masked by impaired consciousness, and in this condition is paroxysmal, tends to be precipitated by excitement, exertion, and by stooping. It is often associated with irritability, nervousness and giddiness.

14. Fibrositis:

This is a recognized cause of headache. Tender local spot should be sought for and aggravation on pressure should not be forgotten.

15. Referred Pain:

Remote visceral lesions may produce referred pain in superficial tissues, innervated by the same segment of the nervous system, such as eye-strain, iritis, glaucoma, middle-ear disease, nasal lesions. teeth, pharynx, tongue, intrathoracic and intra-abdominal visceral lesions. It is pointed out that the

trigeminal nerve is the somatic sensory nerve corresponding to the vagus, by which so many viscera are innervated. Nasal obstruction is a common cause of persistent frontal headache. Occipital headache is often present in cases of cervical fibrositis, spondylitis and osteo-arthritis of the upper cervical vertebrae.

16. Intra-cranial Neoplasm:

Headache is probably mainly due to abnormal tension in the cerebral blood vessels (Northfield, 1938). It is paroxysmal in early stages. It is often described as a throbbing or a "bursting" pain. It occurs chiefly during the night and in the early morning. The patient awakens with a headache lasting a few minutes and then passes off, to recur again the next day, with gradual enlargement of the growth, the headache tends to become more prolonged and may ultimately be continuous. It is always intensified by exertion such as excitement, coughing, vomiting, stooping and straining at stool. It is also influenced by posture, becoming worse when lying down or lying upon one side and may be relieved by sitting up.

17. Diseases of the bones and cranium :

Syphilitic osteitis and the osteitis deformans of Paget should be borne in mind. Headache due to osteitis is of a burning, boring character and is associated with tenderness of the skull, which often feels warmer than normal. Local or general

thickening of the cranium is often present. Radiographic demonstration of the characteristic changes in the bones may be obtained. Craniostenosis due to premature synostosis of the sutures is readily recognized by the abnormal shape of the skull.

18. Neuritis and Neuralgia:

Pain in the head may be due to neuritis or neuralgia of the sensory nerves of the scalp. The supra-orbital, auriculo-temporal, posterior auricular and great occipital nerves may be the site of such process. The pain is usually paroxysmal, radiating along the course of the nerve, which is tender on pressure. Cutaneous hyperalgesia corresponding to the sensory distribution of the affected nerve is usually present. Trigeminal neuralgia in the distribution of the nerve may be due to pressure in its intracranial course by intracranial neoplasm or aneurysm or meningo-vascular syphilis or tabes or its centre fibres may be included in the lesions within the medulla. Thrombosis of the posterior inferior cerebellar artery, syringo-bulbia, and disseminated sclerosis may in this way cause neuralgic pain over the face and scalp.

19. Meningitis:

Meningeal irritation is responsible for some of the severest headaches. It may be due to various forms of meningitis, including Syphilitic Meningitis, or to the presence of non-infective

irritant products such as extravasated blood in contact with the meninges. The pain is constant, severe and throbbing or "bursting" and is usually associated with hyperalgesia of the scalp, and, in the case of acute meningitis with other signs of meningeal irritation, such as cervical rigidity and Kernig's sign.

20. Headache of Vascular Origin:

It can be summarized that the high blood pressure causes paroxysmal throbbing or "bursting" headaches. Intra-cerebral haemorrhage results in the mass of the brain, which leads to headache, if the patient remains conscious. Cerebral embolism and thrombosis cause headache due to oedema of the infected area of the brain. Intracranial aneurysm is rarely large enough to cause increased intracranial pressure before rupture, but it may cause pain in the head by compression of the trigeminal nerve. After rupture, subarachnoid haemorrhage leads to headache by causing both increased intracranial pressure and meningeal irritation.

Changes in the calibre and permeability of cerebral vessels may be responsible for the headaches accompanying numerous toxic states such as severe infections, alcoholic over-indulgence, general anaesthetics, uraemia and diffuse cerebral inflammation, e.g. the various forms of encephalitis.

Migraine is probably also a vaso-motor disorder, and thus, headache may be described hypothetically, as due to vascular dilatation following preliminary constriction.

Arteritis of the superficial temporal artery may lead to headache. The raised intracranial venous pressure may also cause it. When caused by thrombosis of the intracranial venous sinus, its nature is made obvious by the symptoms and signs. If the raised venous pressure is extra-cranial, the source of the headache may often be missed. Severe paroxysmal "bursting" headaches may thus accompany large goitres, intrathoracic neoplasm, aneurysm and chronic emphysema, but at times emphysemal headache occurs with each bout of coughing.

Known mechanisms of headache production:

Wolff (1943) concludes from the comprehensive work that the pain sensitive structures of the head are:-

(1) Of the tissues, covering the cranium, all were more or less sensitive to pain, the arteries being especially so.

(2) Of the intracranial structures, the great venous sinuses and their venous tributaries from the surface of the brain, parts of the dura at the base, the dural arteries and the cerebral arteries at the base of the brain, the fifth, ninth, and tenth cranial

nerves and the upper three cervical nerves were sensitive to pain.

The cranium (including the diploic and emissary veins), the parenchyma of the brain, most of the dura, most of the pia-arachnoid, the ependymal lining of the ventricles and the choroid plexuses were not sensitive to pain.

From the available data, six basic mechanisms of headache from intracranial sources have been formulated. Headache may result from:

(1) Traction on the veins that pass to the venous sinuses from the surface of the brain and from displacement of the great venous sinuses.

(2) Traction on the middle meningeal arteries.

(3) Traction on the large arteries at the base of the brain and their main branches.

(4) Distension and dilatation of the intra-cranial arteries.

(5) Inflammation in or about any of the pain sensitive structures of the head.

(6) Direct pressure by tumours on the cranial and cervical nerves containing pain afferent fibres from the head.

In spite of this some-what exhausting list of the causes of headache and mechanisms, only a brief mention is made of headache in hypertension.

(Wolff (1943), however, has described in detail the many types of headache, met with in hypertensive patients, on a vascular theory. According to him, the headache in many instances is known to

have preceded the onset of the hypertension and in some cases it changed only in intensity with the rise in the blood pressure.

He further states that the high level of blood pressure among hypertensive patients need not be a factor in the production of headache. Nevertheless, some people suffering from hypertension never had a headache until the hypertension became established. He, however, has postulated that a cranial artery only slightly related, for whatever reason, would not distend as much, and possibly not to the point of producing pain, if the blood pressure were low. If, however, the sustained level were raised, distension would be greater and therefore pain might readily follow. In other words, a degree of changes in the contractile state of the arterial wall, compatible with comfort when the blood pressure is average, is associated with pain when the blood pressure is elevated.

This conception is supported by analogy with experimental evidence of histamine headache (Wolff and Pickering(a)). The headache and maximum distension of the cranial arteries occur not immediately after the injection of histamine, when the effect on the contractile state of these vessels is greatest, but some time later, when the blood pressure returns to its initial level. It is at this time that the walls of the cranial artery react to the elevated pressure and the headache becomes associated with a level of blood

pressure, which is ordinarily associated with comfort. The relation of arterial walls is thus seen to be one necessary factor in the production of histamine headache, and the level of the blood pressure, the other. This is a very close analogy to the hypertensive state. During an average or a normal contractile state of the arterial walls, distension does not occur and, correspondingly, there is no headache, but should this contractile state be impaired, as by stress or fatigue, distension and headache follow.

He also states that headache associated with either decreased or increased intracranial pressure, resultsfrom traction or displacement of pain-sensitive intracranial structures, and is independent of generalized changes in intra-cranial pressure.

As hypertension produces changes in the vessels of the body, or is associated with such changes, it seems highly likely, that the headache observed in patients suffering from the many causes of hypertension, has a basis, some alterationin structure or in function of these blood vessels.

An attempt is now made to study the many causes of hypertension in detail and the presence of headache due to these respective causes would be carefully analysed to understand the possible mechanisms of the headache encountered in patients suffering from the various forms of hypertension.

The factors concerned in maintaining the level of the blood pressure in the body and the pathological changes resulting from elevation, and the causes of such elevation of the blood pressure, also would be considered.

Hypertension.

Factors maintaining the blood pressure:

The systolic pressure is directly dependent upon the output of the heart, while the diastolic pressure is directly dependent upon the peripheral resistance of the blood vessels.

Thus there can be two main types of hypertension:

(1) The systolic hypertension where the systolic pressure alone is elevated.

(2) A diastolic hypertension where the diastolic pressure is elevated.

1. Systolic Hypertension:

If one deals first of all with the systolic hypertension, we know that the causes of hypertension when only the systolic blood pressure is elevated are the conditions, where there is an increased cardiac output which results in a systolic pressure of 150 mm Hg. or more and the diastolic pressure is normal or reduced. (90 mm. Hg. or under).

These conditions comprise:-

1. Exercise.
2. Fevers.
3. Hyperthyroidism.
4. Aortic-regurgitation.
5. Arterio-Venous Aneurysm.
6. Atheroma of aorta and great vessels.
7. Complete heart-block

2. Diastolic Hypertension:

Keith (1949) has described concisely the facts upon which the diastolic pressure depends.

Diastolic hypertension is a condition characterised by a diastolic blood pressure which is consistently above normal (i.e. 90 mm Hg. or higher) under basal waking condition. The diastolic pressure is an indication of the state of the arterioles in general at any given time, and the fundamental haemodynamic alteration in diastolic hypertension is an increased resistence to the outflow of blood in some greater or lesser portion of the arteriolar bed. As a result of this increased peripheral resistance the diastolic pressure, which represents the reduced force in the circulation during diastole, is elevated.

The systolic blood pressure is merely a measure of the myocardial response to this increased resistance against which it must work. At the onset of diastolic hypertension in any individual, the diastolic pressure is slightly but definitely raised, but the systolic pressure may be maintained. As the condition persists, the systolic pressure also will be elevated, the normal ratio of systolic

to diastolic pressure in the average case of essential diastolic hypertension (when not in cardiac failure) being 1·7 : 1·0. In the later stages, however, when the hypertensive heart failure has commenced, the systolic pressure falls with the failing myocardium,while the diastolic pressure remains high, giving effect of a "decapitated" hypertension, indicating probably an evil omen, due to failure of the heart to cope successfully with the increased work still being given it to do.

It would appear that in arterial hypertension, it is the diastolic pressure which is by far the more significant of the two.

If we now consider the matter further, and arrive at a clear conception of what is meant by "essential" disatolic hypertension, it would appear that for many years it has been realised that there are numerous conditions which produce an elevated diastolic pressure.

Very clear and concise classification of these conditions have been carefully worked out by Gilchrist (1941) and (Page and Corcoram). A short summary of such causes of raised diastolic pressure may be :

I. <u>Extra-Renal</u>.

A. Neurological.

1. Emotional or psychic states.
2. Space occupying intracranial lesions.
3. Mid-brain and brain-stem lesions.
   (a)poliomelitis, (b) encephalitis,
   (c)trauma.
4. Diencephalic Syndrome.

B. Endocrinal.

1. Supra-renal dysfunction.
   (a) Paroxysmal hypertension (Pheochromocytoma).
   (b) Adreno-genital Syndrome (Cortical Adenoma).
2. Pituitary adenoma.
   (a) Cushing's basophil adenoma.
3. Ovarian Hypofunction.
   (a) Menopausal.
   (b) Ovarian neoplasms.
4. ? Placenta.
   (a) Certain pregnancy toxaemia.

C. Peripheral (Arteriolar Spasm).

1. Toxic.
2. Essential or unknown.

II. Renal Ischaemia:

A. Occlusive Vascular disease.

1. Renal arteriolar sclerosis.
2. Stenosis of a main renal artery.
3. Co-arctation of the aorta.
4. Amyloidosis.
5. Infarction, thrombosis, trauma.
6. Congestive heart failure.
7. Aberrant renal artery.
8. Periarteritis nodosa, disseminated lupus erythematous.

B. Primary renal disease.

1. Chronic glomerulo-nephritis.
2. Chronic pyelo-nephritis.
3. Renal tumours.
4. Polycystic disease.
5. Nephroptosis.

C. Renal Reflux.

Hydronephrosis:- Congenital or acquired.

1. Bilateral:
   (a) Urethral obstruction.
   (b) Prostatic obstruction.
   (c) Vesical neoplasm.
   (d) Pelvic tumours, ? pregnant uterus.
   (e) "Spinal" bladder.

2. Unilateral:
   (a) Impacted calculus.
   (b) Ureteral strictures.
   (c) Sympathetic "Imbalance".

Essential hypertension is frequently met with and it is observed that essential diastolic hypertension accounts for 90-95% of all cases of hypertensive heart disease (White, 1946). It has widespread incidence and manifold symptoms and it presents a mass of material for clinical research more than is provided by any other condition.

It will not be out of place to discuss briefly the factors already known about this condition.

The fundamental cause of this condition is, as we have seen, still unknown, but theories are by no means lacking. Increased peripheral resistance in man can result from heightened vaso-constrictor activity mediated neurogenically or humorally. Neurogenic over-activity of the vaso-constrictor fibres may be induced in various ways, for example, different pathways may be stimulated by a change in the environment (e.g. cold) and psychogenically as in emotional excitement, humoral vaso-constriction may be due to a single pressor substance, or a number of different substances, being circulated through the body to act locally on the arterioles, but the actual site of manufacture of these substances still appears to be unknown, although Goldblatt's (1934) famous experiments on clamping the renal artery of a dog have seriously incriminated the kidney but not finally

accepted as solving the problem.

It is likely that summation of impulses from an ischaemic kidney cause the output of some internal secretion, which obviously by central or peripheral action lead to generalized vaso-constriction; or it may be that the presence of an ischaemic kidney leads to the accumulation or new formation of a substance or by a disturbance of chemical equilibrium between substances normally present in the blood, resulting in a pressor action (Goldering and Chasis, 1944).

Renal denervation fails to relieve ischaemic hypertension:

It has been found experimentally that "renal ischaemic" hypertension cannot be relieved by renal denervation, and can be produced in renal denervated animals. (Page and Corcoran, 1945).

Splanchnicectomy does not relieve ischaemic hypertension:

It has been found that hypertension due to renal ischaemia cannot be abolished by splanchnicectomy and coeliac ganglionectomy (Tigerstedt and Bergmann, 1898).

Suggests a humoral rather than a neurogenic mechanism:

Thus it would appear that it is due to a humoral rather than a neurogenic mechanism and as is borne out by the fact that if a kidney is rendered ischaemic and the renal veins are obstructed, no hypertension results.

Further work by Tigerstadt and Bergmann,(1898),

Houssy, Fasciolo and Taquini (1938). Page and Helmer (1940) and Harrison, Grollman and Williams (1942) have resulted in the finding of a pressor substance (called "Angiotonin" and "hypertensin"), produced by the kidney, and its neutralisation by other(antipressor) substances.

Short-circuiting mechanism :

More recently work of Trueta and his associates (1947) suggests that under certain conditions, the renal cortex is rendered ischaemic by a short-circuiting mechanism whereby the blood from the renal artery by-passes the cortex and travels direct to the medulla via the vasa-recta.

Further experiments on rabbits:

Experiments on rabbits has shown that this mechanism can be initiated by a neurovascular reflex and it is possible that further work on these lines may throw light on some of the problems of essential hypertension.

The most widely held theory at the present time, is that in the hypertensive individual, the arterioles throughout the body have, through some direct toxic or nervous influences, or as a result of inherited vascular-sensitivity, become irritable and pass into a state of vaso-constriction thus increasing the peripheral resistance.

To start with,the increased resistance is symptomless; later the course of events may vary;

the arteriolar spasm may wax and wane and may finally disappear, at times resulting in a spontaneous "Cure". Or as more often occurs, it may become permanent, causing a variety of symptoms and shortening life to some extent. Finally, it may progress with extreme rapidity, causing arteriolar necrosis and the symptom-complex of "malignant hypertension".

The cause of essential diastolic hypertension is, however unknown, as also is the cure.

Pathological Changes in the Body in Hypertension:

Systolic hypertension with normal diastolic pressure is less important clinically than diastolic hypertension. It apparently is the result of arterio-sclerosis and does not give rise to any important degree of heart disease.

Nevertheless, patients with systolic hypertension have been investigated with a view to determine the nature of headache when present in these cases.

An elevation of the diastolic pressure, on the other hand, however, produces definite changes that are encountered in cases of hypertension.

They comprise:-

1. Changes in the heart.
2. Changes in the blood vessels.
3. Changes in the kidney.
4. Changes in the other organs.

(1) Changes in the heart: (Cardiac Lesions).

The pathology of hypertensive heart disease is obvious. Paul White (1946) states that both cardiac and vascular abnormalities in chronic hypertension are primarily but natural responses of muscle to increased work. Hypertrophy of individual muscle fibres of the left ventricle is always present, sometimes to such a degree that the heart is greatly enlarged. A heart weight of about 500 grams (normal = 200-300 grams) is common and in rare cases this may be increased to 750 or even to 1000 grams. With the development of failure dilation appears,

changing the appearance of left ventricular hypertrophy from "Concentric" to "Eccentric". Such left ventricular dilation is followed by dilatation of the mitral valve ring, functional mitral regurgitation, dilatation of the left auricle, and enlargement of the right ventricle and auricle too, if the left ventricular failure lasts long enough. It has been suggested that the primary hypertrophy of the heart muscle begins only after it has been strained or traumatized and somewhat dilated by the early efforts to overcome the effect of the arteriolar constriction.

Since with systolic hypertension, the pulmonary arterial blood pressure usually remains normal (until left ventricle fails), the right ventricle is unaffected early in the disease. Eventually after the left ventricle has begun to fail, the pulmonary engorgement increases, and the right ventricle in its turn, is subjected to considerable strain and begins to enlarge. As a matter of fact the commonest cause of right ventricular enlargement is failure of the left ventricle secondary to systemic hypertension.

There is no actual myocarditis or myocardial degeneration in most cases of uncomplicated hypertensive heart disease, even in the presence of massive hearts with marked congestive failure; some myocardial scarring (fibrosis usually in small area) is, however, not uncommon, even in the absence of coronary disease (Levine V; 1934).

Endocarditis and pericarditis do not occur primarily in this type of heart disease, although endocardial sclerosis, most marked in the left auricle which first bears the brunt of left ventricular failure was found by Levin in all of a series of 27 hypertensive hearts.

2. Vascular changes:-

(a) Aorta: The aorta, normal at first, becomes dilated in older and chronic cases,but never to the degree observed in advanced syphilitic aortitis. Some of the dilatation seen by X-Ray examination is not found post-mortem, since it is temporary, depending on the intra-aortic hydrodynamic state. The vascular dilatation may extend a little into aortic branches especially into innominate and carotid arteries.

(b) Smaller Vessels: Diffuse arteriolar sclerosis is the name given to the lesion in the smaller vessels in hypertension.

This form of arterial degeneration is also called arteriolosclerosis and diffuse hyperplastic sclerosis. The term arteriole is vague and not susceptible of strict definition. In the present connection it is used to indicate the smaller arteries of the viscera, the intimate vasculature, vessel 100 microns in diameter or less. The lesions are not all of one type, but in general they cause thickening

of the wall and narrowing of the lumen. Arteriolar sclerosis may be wide spread, but is most frequent in spleen, pancreas, kidney and adrenal (Boyd, 1950). The arteries involved are of a smaller order than the "small" arteries affected by atheroma, e.g. coronary and cerebral vessels. (Boyd, 1950).

Hypertension and ageing process seem to be the two principal aetiological factors. That hypertension is a causal agent is indicated by the fact that identical lesions are found in the experimental hypersensitive animal. On the other hand similar lesions although usually less pronounced, may be present in persons without hypertension (Boyd) especially in the aged. Hypertension appears to accentuate and speed up a normal wear-and-tear degenerated process.

The distribution of arteriolosclerosis is different in hypertensive and non-hypertensive cases. In hypertension the most marked lesions particularly subintimal hyaline thickening, are those of the small renal vessels; indeed such lesions in the kidney when at all diffuse may be regarded as the hallmark of hypertension. They are also found, in much less degree, in the small vessels of the abdominal viscera, especially the spleen, pancreas and adrenals, (Boyd). In non-hypertensives, on the other hand, arteriolar thickening is found as an ageing process in the abdominal viscera with the exception of the kidneys.

It is in hypertension that the lesions are best developed. Essential hypertension may be divided into so-called benign and malignant forms. The benign form is characterised by a gradual onset and a protracted course. The malignant form, much less common, is frequently of abrupt onset and runs a course measured in months rather than years. It often ends with renal failure (Uraemia), but not necessarily so. The lesions of hypertensive arteriolosclerosis differ in two forms, although the distinction between the two is not always as sharp as indicated. In each form there may be two significant lesions.

The Benign Form:

The characteristic lesions are hyaline degeneration and elastic hyperplasia. Hyaline degeneration, the commonest manifestation of arteriolosclerosis is best seen in smallest vessels, such as the afferent arterioles of the kidney, although not confined to these vessels. In the kidney, it is almost invariably associated with hypertension, whereas in non-hypertensives the kidney is one of the organs least frequently involved. In no other organ is there this constant relationship between arteriolosclerosis and hypertension.

Elastic hyperplasia (elastosis) is most marked in the larger arterioles and medium-sized arteries, but some degree of it can be seen in the smallest vessels.

The Malignant Form:

In the malignant form of hypertension, in which the process has a quickened tempo and the vessels have less time to adopt themselves to increased strain, the characteristic lesions are intimal hyperplasia and arteriolar necrosis. Intimal hyperplasia commonly called productive endarteritis and hyperplastic arteriolosclerosis, is a condition in which the intima of the arterioles and small arteries is uniformly thickened with corresponding narrowing of the lumen.

In arteriolarnecrosis, also called necrotizing arteriolitis, the whole thickness of the vessel wall becomes necrotic and structureless. This is in sharp contrast to the clean-cut smooth appearance of hyaline degeneration, in which sharply-defined nuclei often persist. The necrotic wall often becomes infiltrated with red cells, and haemorrhage is common. Arteriolonecrosis is commonly seen in hypertension complicated by renal failure (Uraemia), and it is possible that the toxic products in the blood may play a part in its production. Arteriolonecrosis is also seen in renal failure produced by experimental methods.

In autopsy material, however, the pathological changes described above are usually combined, for in many cases benign hypertension may have a malignant termination.

3. Changes in the Kidney:

The arteriolosclerotic kidney, producing the renal condition of nephrosclerosis, is the primary contracted kidney, the true granular contracted kidney, what the Germans call "Genuine Schrumpfniere".

Arteriolar nephrosclerosis is a very much commoner cause of renal failure than glomerulonephritis. It is the renal end-result of a condition, which is primarily vascular and not renal.

The effect upon the kidney depends entirely on the extent and intensity of the vascular lesions, and probably on the rapidity with which they develop. With regard to structure, therefore, the kidney may appear normal to the naked eye, only manifesting under the microscope what Moscheowitz calls minimal renal lesions, it may be slightly shrunken or completely contracted and extremely granular, while as regards function, there may be every grade between the normal and the most complete renal insufficiency with uraemia.

In case of benign hypertension: therefore, it will be obvious that the kidney may vary from a condition which, as far as renal function is concerned, is to all intents and purposes normal, to one of profound renal disintegration and break-down.

In the fully developed picture the kidney is small and shrunken, the capsule is adherent to the surface, and the surface itself is finally granular

and mottled owing to an alteration of pale-elevated nodules and red depressed portions. Here and there small cysts may be seen on the surface.

The microscopic examination shows a picture which varies enormously. In one case, apart from the vascular changes there may be hardly any deviation from the normal except for here and there a glomerulus partly or completely fibrosed. In another, great numbers of glomeruli may show profound fibrotic changes, with corresponding changes in the tubules and connective tissue.

The obliteration of many glomeruli is naturally followed by the collapse, atrophy and practical disappearance of large numbers of tubules. This is followed in turn by a great increase in the connective tissue, an increase both apparent (due to general condensation which accompanies the shrinkage) and real. Nothing is more difficult than to determine the exact sequence of events in renal lesions, but it would appear here as if the hypertension was the starting point from which followed in turn the thickening and narrowing of the afferent arteries, the collapse and obliteration of the glomeruli, the atrophy of the tubules and the great increase in the interstitial tissue of the kidney.

The Kidney of Malignant Hypertension:

In most of the cases of essential hypertension, there is no evidence of renal failure for a long

time, although the patient is eventually likely to succumb to uraemia if he has not already died of cerebral haemorrhage or cardiac failure. In a small group of cases, however, acute and progressive renal insufficiency develops at an early date, usually at an early age, i.e. in the thirties and forties. These are the cases of malignant hypertension, and the renal changes have been described as Malignant Nephrosclerosis. This is the original "Combination form" of Volhard and Fahr. The blood pressure is likely to be higher than in benign hypertension, and yet death is usually due to uraemia. The essential difference between the benign and malignant forms is a quickening of the tempo, with increased intensity amd distructiveness of the lesions.

The gross appearance is often that of a smooth kidney of normal or even increased size,owing to the fact that the process has progressed too rapidly for much atrophy to develop. In other cases it is granular and contracted. Not infrequently the two kidneys differ markedly in size. The surface may be covered with haemorrhage, usually large and blotchy, sometimes small and petechial. In the latter case the appearance is that of "flea-bitten kidney".

Microscopically:

The chief additional lesion apart from those described in the benign form is an arteriolonecrosis,

affecting principally the afferent arterioles. Owing to the necrosis haemorrhage may occur either into the glomerular space or into the surrounding tissue. This explains both the haemorrhages seen on the surface and the presence of red blood cells in urine.

4. Changes in the other organs:

Fishberg investigated the arterial lesions in 72 cases of essential hypertension which came to autopsy. "In every instance the minute arterioles of the kidney were affected. The splenic arterioles were affected in about two-thirds of the cases, the pancreatic in about one-half, the hepatic in less than one-third, and the cerebral in about one-fifth. The lesions in these organs, when present, were not nearly so marked as in the kidney. The terminal arterioles of the skin, skeletal muscle, myocardium, lungs, gastro-intestinal tract, and thyroid were very rarely involved and then only to an insignificant extent. The distribution of arteriolosclerosis is different from that of large vessel arterosclerosis; the latter has its site of predilection in the heart, brain and extremities, while arteriolosclerosis is most frequent in the kidney, spleen and pancreas".

The arteriolosclerotic lesions, of both benign and malignant type, are found in several of the abdominal viscera, particularly the spleen, pancreas, the capsule of the adrenal glands and to a lesser degree the liver. Kernohan, Anderson and

Keith have investigated the condition of the arterioles in small pieces of the pectoral muscle removed for biopsy, they compared the diameter of the lumen with the thickness of the wall and came to the conclusion that extreme narrowing of the lumen was much more characteristic of malignant than of benign hypertension. Andrus has attacked these conclusions, and has shown that the wall to lumen ratio depends to a great measure on the degree of contraction of the artery, which is indicated by the tortuosity of the internal elastic lamina. Different segments of the same artery showed different degree of contraction.

The retinal changes in hypertension form an interesting subject, which has been studied recently by Wagener and Keith, Fishberg and Oppenheimer and others. The familiar term albuminuric retinitis is a generic designation for all lesions of the retina in patients with renal disease.
There are three fundamental retinal lesions in hypertension (Fishberg and Oppenheimer):

(1) Arteriolosclerotic retinopathy.
(2) Malginant hypertensive neuroretinitis.
(3) Choked disc due to increased intracranial tension from oedema of the brain.

1. <u>Retinal arteriolosclerosis:</u>

The least significant of these three, for it does not necessarily indicate any renal insufficiency, the patient often living many years and dying

of cardiac failure, coronary occlusion, or cerebral haemorrhage. The arteries are sclerosed and show white lines, there may be some irregular constriction of the vessels, but there is complete absence of papilloedema. Small haemorrhages may be present in the retina together with white spots of sharp outline ("hard" spot), some of which may form a star-shaped figure around the macula.

2. Malignant hypertensive neuro-retinitis:

Papilloedema is usually the first change and is the distinguishing feature of the condition. Indeed it is the presence or absence of papilloedema which is the distinguishing mark between the malignant and benign forms of hypertension. The previously described retinal changes are again present, namely, haemorrhages and white spots, but the latter are of the "soft" or "cotton-wool" variety,although hard spots may also be present. Arteriolosclerosis may or may not be present, but the arteries are always markedly narrowed owing to the functional vasoconstriction. The retinal lesions appear to be produced by arterial spasm.

3. Choked disc:

Would appear to be the result of cerebral oedema.

Hypertension has been classified on a

physiological and pathological basis and the effects of high blood pressure on organs in the body have been discussed at length. It remains now, to analyse these known facts and to see, what factors, if any, there are in common between hypertension and the recognized causes of headache.

The Factors in common between Hypertension and known causes of Headache:

The causes of headache may be summarized again.

Headache as:

1. Vascular origin.
2. Disease of the Bones of the cranium.
3. Neuritis and Neuralgia.
4. Meningeal Irritation.
5. Referred Pain.
6. Raised intra-cranial Pressure.
7. Intra-cranial Abscess.
8. Trauma.
9. Lowered Intra-cranial pressure.
10. Psychological.

Each cause will be analysed in relationship to the presence in the body of hypertension.

1. Vascular Origin:-

It is known that changes in the calibre of blood vessels result in headache (Wolff). Retinal examination of some patients with hypertension show spasm of the arterioles. The cold-pressor test and the Sodium Amytal test, when performed in patients with hypertension, suggest an unstable state of the blood vessels and further suggest that vascular spasm in these patients is a common occurrence. It would thus appear that one cause of headache in hypertension may be an unstable state of the blood vessels resulting in changes in calibre of the intracranial vessels.

2. Disease of the Bones of the Cranium: and other covering structures:

It is unlikely that hypertension would result in any pathological change in bone. This cause, therefore need not be considered.

The retinal changes in hypertension produce impairment of vision. This impaired vision causes the patient to "strain" when reading or indeed,using the eyes at all. This "strain" is associated with prolonged spasm of the frontalis muscle and this may produce headache.

3. Neuritis and Neuralgia:

The renal impairment which we know is associated with some types of hypertension may result in headache caused by the "Uraemic State" producing neuritis. This, however, is unlikely.

4. Referred Pain:

It is unlikely that referred pain plays any part in the headache of hypertension. If, however, such conditions as sinusitis or occipital fibrositis or cervical osteoarthritis are present, then any headache, due to hypertension, may be felt over these already unduly sensitive structures.

5. Meningeal Irritation:

Hypertension could only produce headache by meningeal irritation if:

(a) The meninges were irritated by haemmorhage.
(b) The meninges were irritated by urea or other metabolic products which were not secreted by the kidney.
(c) If as a result of hypertension, there is cerebral oedema the stretching of the meninges would occur and perhaps produce headache.

6. Raised Intra-cranial Pressure:

We know that there is an important general cause of headache. In certain cases of hypertension there is papilloedema. This would suggest that in these cases of hypertension, there is a raised intra-cranial pressure. We also know that some types of hypertension may arise from intra-cranial lesions (Gilchrist) e.g. cerebral tumours, and mid-brain and brain-stem lesions. These conditions may elevate the intra-cranial pressure resulting in headache as well as hypertension.

7. Intra-cranial Abscess:

As a cause of headache in hypertension need not be considered.

8. Trauma:

Is unlikely as a cause of headache in hypertension.

9. Lowered intra-cranial pressure:

It is difficult to visualize this state arising in patients suffering from hypertension unless there is some sudden change in the haemodynamics of the circulation.

10. Psychological:

This may play a part in the cause of headache in hypertension. The patient may be introspective, be unduly nervous and knowing he is a chronic sufferer complain of headache to attract attention and sympathy.

The above suggestions would suggest that the many possible explanations of headache in patients suffering from the many types of hypertension may be therefore:-

1. Changes in the calibre of the cerebral vessels.
2. Spasm of the frontalis muscle due to impaired vision resulting from retinal changes.
3. Raised intra-cranial pressure.
4. Lowered intra-cranial pressure due to some alteration in the haemodynamics of the circulation.
5. Psychological.
6. Perhaps meningeal irritation.
7. Perhaps but unlikely a "neuritis" associated with changes in the blood chemistry consequent to renal failure.
8. Perhaps referred pain to some other pain sensitive area.

Although the above causes of headache in hypertensive patients are suggested, nevertheless we know that patients with gross hypertension may never complain of headache, yet the patients in whom hypertensive state is not well advanced, complain of intractable headache. The headache may vary in each individual in site, type of pain and time of onset. It would thus appear that other unknown factors, in addition to the above, play a part in the mechanism of this type of headache.

CASE MATERIAL and METHODS of INVESTIGATION.

In this series, 82 patients suffering from headache due to hypertension were investigation and their case histories have been critically surveyed from the clinical aspect. Symptoms of headache were studied in detail and an attempt has been made:

(1) to assess the incidence of headache in various forms of hypertension.

(2) to determine the character of this symptom in different varieties of hypertension.

(3) to determine any precipitating factor in headache production in these cases with a view to prevent its occurrence.

(4) to correlate the severity and incidence of headache with other associated changes in the body resulting from the Hypertension.

(5) to determine from this information, a possible mechanism of headache production.

(6) to give an indication of the prognostic significance of headache.

(7) to discuss the methods of treatment adopted in these cases.

It is recognized by many workers, that of the two main types of hypertension, Systolic and diastolic, it is the latter, which is encountered more frequently, that produces most symptoms and has a graver prognosis. Indeed, some clinicians dismiss systolic hypertension relatively as of no importance. Diastolic hypertension will therefore, be discussed first of all.

As mentioned earlier, by diastolic hypertension we mean a condition characterised by a diastolic pressure of over 90 mm Hg. under basal waking conditions. The Sodium Amytal test (Learmonth,1947) was performed in some cases, wherever applicable, to determine the presence and persistence of such an elevated blood pressure.

Methods and Standards adopted in the examination of these Patients:

In the examination of these patients, suffering from headache, the questionnaire suggested by Symond (1937) was adopted; for in the author's opinion, it cannot be bettered. Every patient was therefore, asked the following questions :-

(1) What is the nature of pain ?

(2) What parts of the head are involved ?

(3) Is the pain continuous or intermittent, if the latter, how frequent are the attacks ?

(4) How long the attacks last.

(5) Does the attack occur at any time or particular time of day ?

(6) What factors, if any, is the attack provoked or aggravated ?

(7) What are the associated symptoms ?

(8) Has the development been immediately preceded or accompanied by any head injury preceding the attacks ?

(9) What gave you relief ?

(10) Inspection of the head by sight and touch was carried out in every case for clinical information and psychological reasons.

Headache, when present in these cases, has been provisionally divided into mild, moderate and severe with a view to indicate the degree of severity.

Headache is a subjective symptom and is therefore difficult to classify with accuracy. It varies in degree from patient to patient depending on many factors. Indeed the degree may vary in the same patient. Some patients suffer from two or more types of headache and when an attack begins they may not be able to indicate its character until it is well developed. (Alvarez, 1940).

From the patient's description, and considering the age, sex, and type of individual, an attempt was made to classify the severity of the headache into the following degrees:-

(a) Mild.
(b) Moderate.
(c) Severe.

Our criteria for grading:

(a) A mild headache being one, which did not inconvenience the patient,but allowed him to continue his normal activity and did not require any therapy.

(b) A moderate headache is one, where the patient took some drug to obtain relief or was associated with failure of concentration.

(c) A severe headache being one which incapcitated the patient and required medication for relief.

In these cases, the cardiac efficiency has been graded in the usual manner viz:-

	Cardiac Efficiency.
(a) Normal	No cardiac symptoms whatever.
(b) Grade I.	Dyspnoea only on severe exertion.
(c) Grade II.B	Dyspnoea on mild exertion.
(d) Grade II. B	Dyspnoea on slightest exertion with or without oedema - early cardiac failure.
(e) Grade III.	Dyspnoea at rest - marked cardiac failure.

In addition, as dyspnoea was present in almost all cases, further factors needed to be considered.

They are:

(1) Symptoms of dyspnoea or pain.

(2) Physical and clinical signs, such as tachycardia, arrhythmias, gallop rhythm, accentuation of the second pulmonary sound, and basal crepitations.

(3) Radiology of the heart and electrocardiographic examination revealed enlargement of the left ventricle in all cases,and therefore, it was of little value in assessing the cardiac grading.

Retinal grading:

The classification of Wagener and Keith (b) was employed in this investigation.

The standards for each grade are as follows :

Grade 0 : Normal

Grade 1 : Slight irregularity of the arterioles with patchy narrowing of the lumen and no other abnormality.

Grade II : Grade I. findings, which, in addition, arterio-venous "nipping" where those vessels cross one another.

Grade III : Grade I. and grade II. findings, with haemorrhages and exudates: i.e. Hypertensive retinitis.

Grade IV : Papilloedema, with all previous grades of abnormality or with only severe arteriolar spasm.

Estimation of Co_2 combining power of the blood: This was estimated according to the method of Van Slyke.

Renal function tests:

Of the many tests of renal efficiency, the one that was used mostly in this series of cases was Calvert's urea concentration range. (Dunlop et al.). In one patient, a boy of 16 years, the Van Slyke urea clearance was employed, for in this case albuminuria was discovered on a routine school medical examination.

Blood Urea Nitrogen : Throughout this series, it was considered to be normal if it was in the range of 10-20 mgm%.

In all these cases, other causes of headache such as migraine, primary refractory errors and sinusitis were excluded by routine examination.

Case Material:

As far as possible as many causes of hypertension corresponding to the classification of Gilchrist (1941) have been included in this series of study.

The cases in this series have been collected from the medical practice in India, the medical wards of a general hospital in India and in the Royal Infirmary of Edinburgh.

They are divided into the following groups, according to the classification of hypertension, devised by Gilchrist:-

I. Diastolic Hypertension:

1. Extra Renal :

(a) Peripheral: (arteriolar spasm)		
1 "Essential Hypertension"	Cases	15
(b) Neurological:		
1 Space-occupying intra-cranial lesion"	"	4
2 Subarachnoid haemorrhage	"	5
(c) Endocrinal		
1 Cushing's Syndrome	"	2
2 Menopausal	"	8

2. Renal Ischaemia :

(a) Primary renal disease		
1 Chronic glomerulo-nephritis	"	10
2 Chronic pyelo-nephritis	"	1
(b) Occlusive vascular disease		
1 Amyloidosis	"	3
Total Cases of Diastolic Hypertension		48

2. Hypertension affecting the Systolic pressure only:

1.	Thyrotoxicosis	Cases	10
2.	Aortic-regurgitation	"	10
3.	Atheroma of aorta and great vessels	"	10
4.	Complete heart-block	"	4
	Total cases of Systolic Hypertension -		34

DIASTOLIC HYPERTENSION.

A. Essential Hypertension.

It is well known that although patients suffering from essential diastolic hypertension may live for many years, provided they do not succumb to any other disease, the effects of this hypertension are ultimately seen. The prolonged peripheral resistance, resulting in the elevated blood pressure, produces cardiac hypertrophy and permanent changes in the blood vessels. These latter changes - i.e. arteriolosclerosis in benign hypertension, or if the tempo of the disease is increased, arteriolonecrosis in malignant hypertension result in changes in the organs throughout the body. These effects have been discussed at length previously but will be summarized again briefly ::-

(1) Cardiac hypertrophy, cardiac dilatation and cardiac failure perhaps with coronary insufficiency.

(2) Benign or malignant nephrosclerosis with renal insufficiency.

(3) Generalized vascular changes resulting at first in Vasospasm and later permanent damage, arteriolosclerosis and arteriolonecrosis.

(4) Various retinal changes.

(5) Generalized hypo function of the many organs of the body.

The cases of essential hypertension which are described fully in the appendix are analysed as follows:-

Key to Table I., IA., IB.

F	=	Frontal.
O	=	Occipital.
M	=	Moderate.
S	=	Severe.
E.	=	Enlarged.
A	=	Acid.
A.S.M.	=	Aortic Systolic Murmur.
M.S.M.	=	Mitral Systolic Murmur.
A_2++	=	Second aortic sound accentuated.
M_1++	=	First mitral sound accentuated.
P_2++	=	Pulmonary second sound accentuated.
L.V.E.	=	Left ventricular enlargement.
L.A.D.	=	Left Axis deviation.
+	=	Present.
-	=	Absent.
R	=	Relief.

Analyses of cases of Essential Hypertension.

Table I.

Sex	Male	Male	Male	Male	Male	Male	Male
Age	51	66	71	53	61	38	69
Weight	10 st.12½lbs.	10 st. 6 lbs.	8 st. 9½ lbs.	-	11 st. 5 lbs.	16 sts.	14 st.10 lbs.
Habit: Smoking	+	+	-	+	++	+	+
Alcoholic	-	-	-	+	++	-	+
Family History	Not relevant	Not available	Not relevant	Not relevant	Not relevant	Nil.	Not relevant.
Previous Illness.	None.	Not relevant	Not relevant	Not relevant	Not relevant	Nil.	Not relevant.
Presenting Symptoms.	Tiredness 18 months. Tightness in chest 2 months.	Intermittent Claudication 5 years. A few weeks of Nocturnal Dyspnoea.	Dyspnoea 5 years.	Coma.	Epistaxis 8 years. Dyspnoea 1 year.	Dyspnoea 1 year.	Rectal bleeding one day. Dyspnoea - 1 year.
Headache:	-	+	+	+	+	+	+
Duration	-	6 months	-	6 months	1 year	6 months	1 year.
Site	-	F.	-	F.	O	F.	O
Character	-	T.	-	T.	T.	T.	T.
Onset	-	In morning	-	In morning	In morning	In morning	Any time.
Severity	-	M.	-	S.	M.	M.	M.
Precp.Factors	-	Rising from bed.	-	Rising from bed.	Rising from bed.	Rising from bed.	Waking, straoning, de nevo.
Ass.Symptoms	-	-	-	-	Occasional Epistaxis	-	-
Duration of Attack	-	Up to ½ hour	-	Up to	1 hour	Up to 1 hr.	Several hours
Relief	-	Rest Luminal "head-up" bed	-	No relief	Epistaxis Venesection "head-up" bed	Rest Luminal "head-up"bed	Rest Aspirin "head-up" bed
B.P.	A D						
Systolic	260 160	240 240	220 190	240	220 180	160 150	210 210
Diastolic	160 110	130 130	100 100	120	120 110	100 100	120 120
Heart:	E A.S.M. A_2 ++	E A_2 ++	E A_2 ++ P_2 ++	E	E A.S.M. A_2 ++	Normal	E
X-Ray	L.V.E. Calcified Aorta.	L.V.E.	L.V.E.Silicosis Calcified Aorta	-	L.V.E. Unfolding Aorta.	L.V.E.	-
E.C.G.	L.A.D.	L.A.D.auricular fibrillation.	L.A.D.	-	L.A.D.	Normal	-
Basal Crepitation	-	Few	Few	Few	Few	-	-
Fundi	I.	III.	I.	IV.	III.	I.	I.
Blood Urea Nitrogen	15 mgm %	20 mgm %	-	-	30 mgm %	18 mgm %	24 mgm %
Urea Range	Sp.gr. Gm/100 c.c.	-	-	-	-		-
Con.	1015 - 3.2	-	-	-	-	1020 - 4	
Dil.	1010 - 0.2					1010 - 0.8	
Sodium Amytal Test	220 - 110 / 110 80	-	-	-	-	-	-
Urinary Vol. in 24 hrs.in c.c.	1550	1500	1600	2400	2800	2000	1900
Reaction	A	A	A	A	A	A	A
Sp. gr.	1016	1018	1012	1010	1018	1020	1016
Albumin	-	-	-	+	Trace	-	Trace.
Sugar	Trace	-	-	-	-	-	-
Casts	-	-	-	Hyaline	Hyaline	-	Hyaline
Treatment	Rest Phenobarbitone. Diabetic Treatment	Rest Phenobarbitone. Aminophylline.	Rest Phenobarbitone. Penicillin therapy. Breathing exercises.	Rest Symptomatic	Rest Phenobarbitone. Venesection	Rest Phenobarbitone. Dietetic measures.	Rest Phenobarbitone.
Result	R.	R.	R.	Died.	R.	R.	R.

Table IA.

Sex	Female	Female	Female	Male
Age	76	72	56	53
Weight	9 st.	-	10 st. 4 lbs.	8 st. 5 lbs.
Habit: Smoking	-	-	-	+
Alcoholic	-	-	-	+
Family History	Mother - Cerebral Haemorrhage	Not available.	Brother - Hypertension	Sister - Coronary Thrombosis.
Previous Illness.	Relevant.	-	Not relevant.	Not relevant.
Presenting Symptoms	Collapsed. one day.	Hemiplegia - 2 days. Dyspnoea - 2 years.	Dyspnoea - 5 years Cardiac Asthma 4 weeks.	Haemetemesis. Peptic Ulcer.
Headache:	+	+	+	-
Duration	1 year	2 year	10 years.	-
Site	O	O	F.	-
Character	T.	T.	T.	-
Onset	In morning	Present during day.	In morning	-
Severity	Mild	M.	M.	-
Precp.Factors	Rising from bed	Worse on rising	Rising from bed	-
Ass. Symptoms	-	Giddiness	-	-
Duration of Attack	Not known.	Several hours	Up to ½ hour	-
Relief	Rest Phenobarbitone "head-up" bed	No relief	Rest "head-up" bed	-
B.P. Systolic	160 160	200	180 180	195 200
Diastolic	110 100	110	100 100	110 110
Heart	E, A_2++	E. A_2++	Clinically no E.	Clinically no E. M_1 ++ A_2 ++
X-Ray	L.V.E. Calcified Aorta	-	L.V.E.	L.V.E. Unfolding aorta.
E.C.G.	Normal	-	L.A.D.	L.A.D.
Basal Crepitation	-	-	Few	-
Fundi	I.	III.	I.	I.
Blood Urea Nitrogen	16 mgm.%	80 mgm.%	25 mgm.%	15 mgm.%
Urea range	Sp.gr. Gm/100 c.c.			
Con.	1030 - 4	-	-	1020 - 3
Dil.	1006 - 0·4			1006 - 0·7
Sodium Amytal Test	160 150 / 100 90	-	-	-
Urinary Vol. in 24 hrs.in c.c.	2100	3000	1500	1600
Reaction	A.	A.	A.	A.
Sp. gr.	1020	1010	1016	1016
Albumin	-	+	+	-
Sugar	-	-	-	-
Casts	-	Hyaline	Hyaline	-
Treatment	Rest Phenobarbitone	Rest Symptomatic	Rest Phenobarbitone Aminophylline	Rest Morphine Phenobarbitone Ulcer diet
Result	R.	Died	R.	R.

Table IB.

Sex	Male	Male	Male	Male
Age	67	48	40	47
Weight	12 st. 13 lbs.	12 st. 2 lbs.	9 st. 5 lbs.	10 st. 10 lbs.
Habit: Smoking	+	+	+	+
Alcoholic	+	-	-	+
Family History	Not relevant	Mother - Apoplexy	Not relevant	Not relevant
Previous Illness	Not relevant	Nil	Nil	Not relevant
Presenting Symptoms	Dyspnoea - 3 wks Nocturnal frequency of urine 3 months	Dyspnoea - 1 week Cardiac Asthma 1 week	Epistaxis - 3 days Dyspnoea - 3 years Cardiac Asthma 3 weeks.	Dyspnoea - 3 months Precardial pain - 3 months.
Headache	+	+	+	+
Duration	13 years	1 year prior to Sympathectomy	6 months	3 months
Site	O	F	F	F
Character	T	T	T	T
Onset	In morning	In morning	In morning	In morning
Severity	M	S	S	M
Precp. Factors	Rising from bed	Rising from bed	Rising and straining	Rising from bed
Ass. Symptoms	-	-	-	-
Duration of Attack	1 hour	Several hours	Several hours	2 - 3 hours
Relief	Rest Phenobarbitone	Sympathectomy	Rest Epistaxis "head-up" bed	Rest Pethidine "head-up" bed.
B.P.				
Systolic	180 / 180	208 / 240	220 / 200	170 / 150
Diastolic	100 / 100	130 / 130	110 / 110	100 / 100
Heart	E M.S.M. A.S.M., A_2++	E A_2 ++ 4th sound	E A.S.M. A_2++, 4th sound	E M_1 ++ A_2 ++
X-Ray	L.V.E.	L.V.E. Unfolding Aorta	L.V.E. Unfolding Aorta	Slight L.V.E. Slight unfolding aorta
E.C.G.	L.A.D.	L.A.D.	L.A.D.	L.A.D.
Basal Crepitation	Few	Few	Few	Few
Fundi	II.	II.	III.	I.
Blood urea Nitrogen	15 mgm.%	18 mgm.%	58 mgm.%	16 mgm. %
Urea Range Sp.gr. Gm/100 c.c.				
Con.	-	1020 - 3·5	1018 - 2	1020 - 2·6
Dil.		1005 - 1	1003 - 1	1008 - 0·6
Sodium Amytal Test	-	208 / 130 - 208 / 130	-	170 / 100 - 140 / 95
Urinary Vol. in 24 hours in c.c.	1600	1660	2000	1500
Reaction	A	A	A	A
Sp. gr.	1013	1020	1018	1020
Albumin	-	-	Trace	-
Sugar	-	-	-	-
Casts	-	-	Hyaline	-
Treatment	Rest Phenobarbitone	Rest Amenophylline Phenobarbitone.	Rest Phenobarbitone 1·V·1 Sucrose Aminophylline	Rest Phenobarbitone Pethidine
Result	R	R	R	R

Comparison of Patients with and without Headache in Essential Hypertension.

Table II.

	With Headache.			Without Headache.		
Number.	12 (7 F. 5.0)			3		
Males	9			3		
Females	3			0		
Average Age.	57.7 years.			58.3 years.		
Average Weight.	11 st. 10 lbs.			9 st. 4 lbs.		
Average age - Males	54.3 years			58.3 years		
" " Females	68 years			-		
Habit : Smoking	9			2		
Alcoholic	5			1		
Family History of Hypertension:						
(a) Males.	1			1		
(b) Females.	2			-		
Average Weight: (a) Males	12 st. 3 lbs.			9 st. 4 lbs.		
(b) Females	9 st. 9 lbs.			-		
Blood Pressure:	Admission	Discharge	Lability	Admission	Discharge	Lability
(a) Average Systolic	210	194	16	225	180	45
(b) Average Diastolic	112	110	2	123	106	17
Clinical Enlargement of Heart:	10 cases - (1)Apex beat 10 cases (2) A.S.M. 3 " (3) A_2 ++ 8 "			2 cases - (1)Apex beat 2 cases (2) A_2 ++ 3 " (3) A.S.M. 1 case		
X-Ray of Heart :	9 cases X-rayed L.V.E. in 9 cases Unfolding Aorta in 3 cases			3 cases X-Rayed. L.V.E. in 3 cases. Unfolding Aorta in 3 cases.		
E.C.G.	9 cases L.V.H. in 7 cases.			3 cases.- L.V.H. in 3 cases.		
Urine: (1) Reaction	Acid.			Acid.		
(2) Average sp. gr.	1016			1015		
(3) Albumin	6			-		
(4) Casts	6 (Hyaline)			-		
Fundi: Grade I.	5 cases.			3		
Grade II.	2			-		
Grade III.	4			-		
Grade IV.	1			-		
Average Blood Urea N_2	29 mgm %			15 mgm %		
Average Urea - range	Sp. gr. Gm/100 c.c.			Sp. gr. Gm/100 c.c.		
Conc.	1021 - 3.2			1017 3.1		
Dil.	1006 - 0.7			1008 0.45		
Sodium Amytal Test.	Done in 3 cases - Average Systolic fall 13 mm Hg. Average Diastolic fall 5 mm Hg.			Done in 1 case - Systolic fall 10 and Diastolic fall 30 mm Hg.		
Result : Relief of Headache:	8 cases			-		
No relief	2 cases			-		
Dead	2 cases			-		

Key to Table II.

F	=	Frontal
O	=	Occipital
A.S.M.	=	Aortic Systolic Murmur.
A_2++	=	Aortic Second Sound accentuated.
L.V.E.	=	Left Ventricular enlargement.
L.V.H.	=	Left Adis deviation.

Results of Table II.

(1) Incidence of headache:

12 patients out of 15 had headache - 75 %
3 patients out of 15 had no headache 25%
Of 12 patients with headache 7 patients had frontal headache and 5 had occipital headache.

(2) Age-incidence :

There is no significance in the age distribution except that the females tend to be older than the male.

(3) Weight :

Males with headache are significantly heavier than the males with no headache.

(4) Habits :

75 % of those with headache smoked.
41 % of those with headache were alcoholic.

(5) Family history :

Greater family history in females with headache than males with headache, otherwise there is no difference between the two groups.

(6) Blood pressure :

There is no significant difference in systolic and diastolic blood pressure in the two groups on admission, but at the time of discharge, there is a significant difference in the fall in systolic and diastolic pressure in the group with no headache compared to the group with headache. (45 to 16 and 17 to 2 mm Hg.).

(7) Heart:-

There is no difference in the two groups in examination of the heart, clinically, radiologically or electro-cardiographically.

(8) Urine :

There is no significant difference in the specific gravity of the urine or the urea-range in the two groups, 50 % of patients with headache had albuminuria. No patient without headache had albuminuria. This is significant.

(9) Retinal changes :

Fundal changes were present in the three cases without headache. This change was Grade I. All 12 patients with headache had fundal changes:-

Grade I. in 5 cases.

Grade II. in 2 cases.

Grade III. in 4 cases.

Grade IV. in 1 case.

This is again significant.

(10) Blood urea Nitrogen :

The blood urea nitrogen was normal in the group with no headache.

It was twice the normal value in the group with headache. This is significant.

(11) Sodium Amytal Test :

It was performed on too few patients to have any value. It would suggest, however, that in the group with headache the blood pressure does not fluctuate.

(12) It can be seen that the headache falls into two groups. (1) Frontal and (2) Occipital.

Table III. compares the clinical features of these two groups.

Analysis and Comparison of Frontal and Occipital Headache in Essential Hypertension.

Table III.

	Frontal.	Occipital.
Number	(a) Total. 7 out of 15. (b) Headache 7 out of 12 - 58%	(a) Total. 5 out of 15. (b) Headache 5 out of 12 - 42%
Sex:	6 M. : 1 F.:	3 M.: 2 F.
Average Age: " " (a) Males (b) Females	50 years. 49 years. 56 years.	69 years. 66 years. 74 years.
Average Weight: " " (a) Males (b) Females	9 st. 12 lbs. 11 st. 11 lbs. 10 st. 4 lbs.	12 st. 13 st. 9 st.
Severity of Headache: Mild Moderate Severe	0 4 3	1 4 -
Duration of Headache: Duration of Attack: Onset: Character: Relief of Headache	23 months. ½ hour - several hours. In morning in all cases. Throbbing. "Head-up" bed 5. Rest and Luminal 2. Epistaxis 1 Sympathectomy 1 Pethidine 1 No relief 1 Nothing taken 1	43 months. 1 hour - several hours. In morning 4 cases; anytime in one. Throbbing. "head-up" bed 3. Rest and Luminal 2. Epistaxis and Venesection 1 Aspirin 1 No relief 1
Average B.P. (a) on Admission	202/113	194/112
(b) on Discharge	200/113	186/108
Average B.P. in mild cases	-	160/110
in moderate cases	188/108	202/112
in severe cases	223/120	-
Number of fundal changes Mild Moderate Severe	7 - 1 in 3, III in one II in 1, III in one, IV in one	5 I in one. I in one, II in one, III in two.
Cardiac Efficiency: (Mild Headache (Moderate (Severe	- Normal in 1; grade I in 3. IIA in 1; IIB in 1.	Normal. Normal in 2, grade I in 2.
Blood Urea Nitrogen: (Mild Headache (Moderate (Severe	- 21 mgm %. 35 mgm %.	16 mgm %. 37 mgm %. -
Urinary Findings: (Vol. Mild (Sp. gr. (Alb. (Casts.	- - - -	2100 c.c. 1020 c.c. - -
(Vol. Moderate (Sp. gr. (Alb. (Casts	1637 c.c. 1016 + in one case. + in one case - hyaline.	2325 c.c. 1015 + in 3 cases. + in 3 cases - hyaline.
(Vol. Severe (Sp. gr. (Albumin (Casts	2020 c.c. 1016 + in 2 cases. + in 2 cases.	
Mortality:	One. (Cerebral Haemorrhage)	One. (Uraemia).

Key to Table III.

M. = Male.
F. = Female.

Preliminary Conclusions from Tables I., II. and III.

The analysis of these patients with essential diastolic hypertension show that :-

1. Incidence of headache :

75 % of the patients with essential hypertension had headache. This is statistically significant.

2. Sex incidence of patients with headache :

Of these 75 % were males and the remaining 25% were females.

3. Age incidence of patients with headache:

The average age of the patients with headache in essential hypertension was 57.7 years. The male patients were younger than the females. The average age for men being 54.3 years and for women 68 years.

The average ages of men and women with frontal headache was 49 years and 56 years respectively. The incidence of frontal headache, therefore occurring in an older age group in women.

The average age of the patients with occipital headache was 66 years for men and 74 years for women. Occipital headache, therefore occurs in an older age group than does frontal headache in both men and in women. Both frontal and occipital headache occur in an older age group in women than in men.

(4) Average weight of patients with headache:

The patients in this series, suffering from essential hypertension, with headache were heavier than the patients who experienced no symptoms of headache.

(5) Habit :

75% of those with headache smoked.

41% of those with headache were alcoholic.

(6) Family history of patients with headache:

Only three patients in this series of patients with headache gave a family history of hypertension. This fact alone is not significant. These three cases comprised two females and one male.

(7) Characteristics and precipitating factor of headache:

(a) The occurrence of the headache in these cases, 91% on rising in the morning. The remaining 9% had a headache at any time.

(b) Frontal headache was commoner than occipital headache.

(c) The character of the headache was the same in every case, being throbbing in nature.

(d) The duration of the attack varied from half an hour to several hours in the cases of the frontal headache and from one hour to several hours in the cases with the occipital headache.

(e) The severity of the headache was different in the two groups of cases. Of the seven patients with frontal headache, four patients had headache which was classified as moderate (see page 41 for severity) and three patients complained of headache which was classified as severe.

In the occipital group, however, five patients complained of headache. One patient had a headache of mild severity and the remaining four were classified as of moderate severity.

(f) No relief was obtained in 16% of these cases.

(8) Liability of Blood-pressure :

When the liability of the blood-pressure is compared in patients with essential hypertension with and without headache, it was found that the average variation in blood-pressure in the group with headache was 16% mm Hg. systolic and 2 mm Hg. diastolic, while in the group without headache, the corresponding variation was 45 mm Hg. systolic and 17 mm Hg. diastolic.

These figures are definitely significant and show that in the group with headache, there is very little if any variation in blood pressure.

(9) Cardiac efficiency :

The cardiac efficiency in patients with frontal headache was as follows :-

Normal	in one case.
Grade I.	in three cases.
Grade IIA	in one case.
Grade IIB	in one case.

The cardiac efficiency of only six patients out of the seven with frontal headache could be assessed, as the seventh patient was admitted in coma and no history was available. While in patients with occipital headache, the cardiac efficiency was normal in three patients and Grade I. in two cases.

(10) Renal functions:

50% of these patients with headache had impaired renal function. This incidence of impaired renal function was higher in the group with occipital headache being 60%, while in the group with frontal headache, the incidence was only 42%.

(11) Retinal changes :

The retinal changes were more marked in patients with headache in this series than in those without headache. This difference is very striking and can be seen in Table II.

(12) Mortality:

Two patients in this series died, one patient with frontal headache had a cerebral haemorrhage and one patient with occipital headache developed uraemia and died from hypostatic pneumonia.

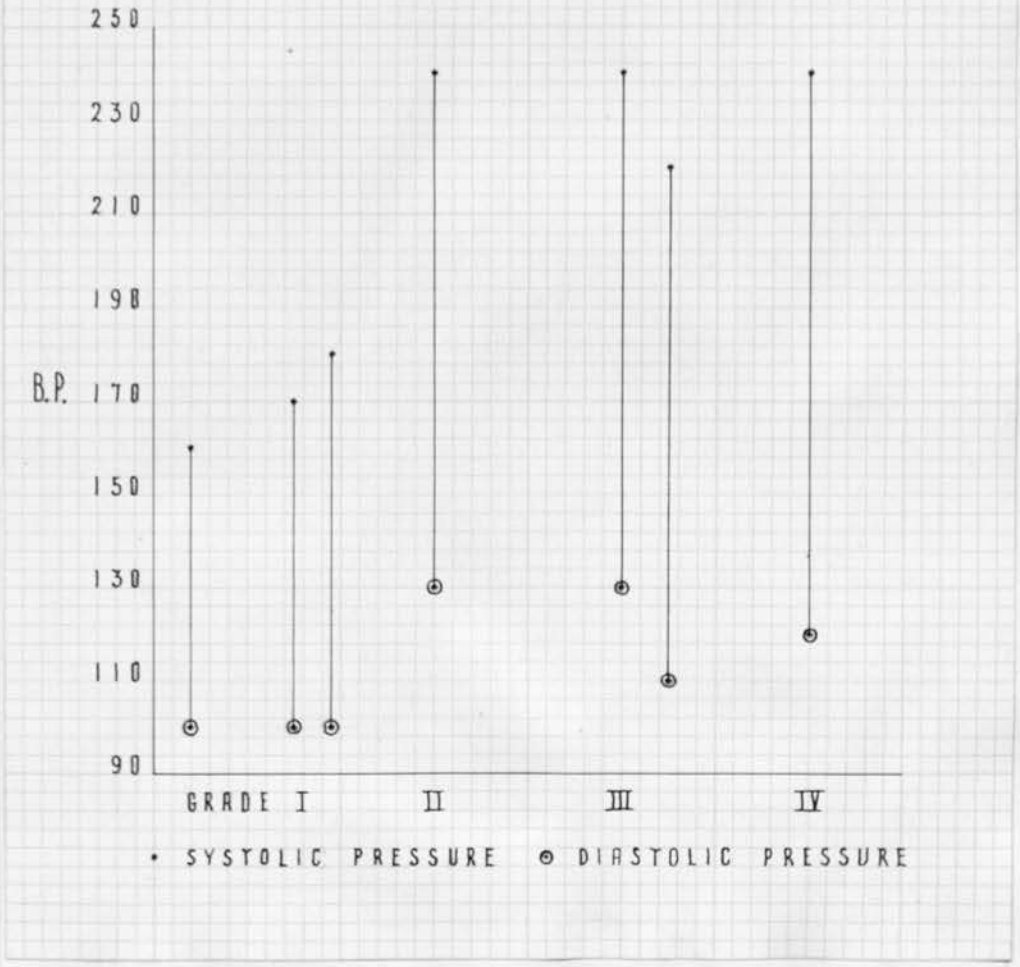
Relationship of Pathological and Biochemical Changes with the Clinical Features of the Headache.

A. FRONTAL HEADACHE.

1. B.P. levels and fundal changes.

The relationship between the levels of systolic and diastolic pressure and the fundal changes in these patients with frontal headache is seen in the following diagram:

Diagram I.

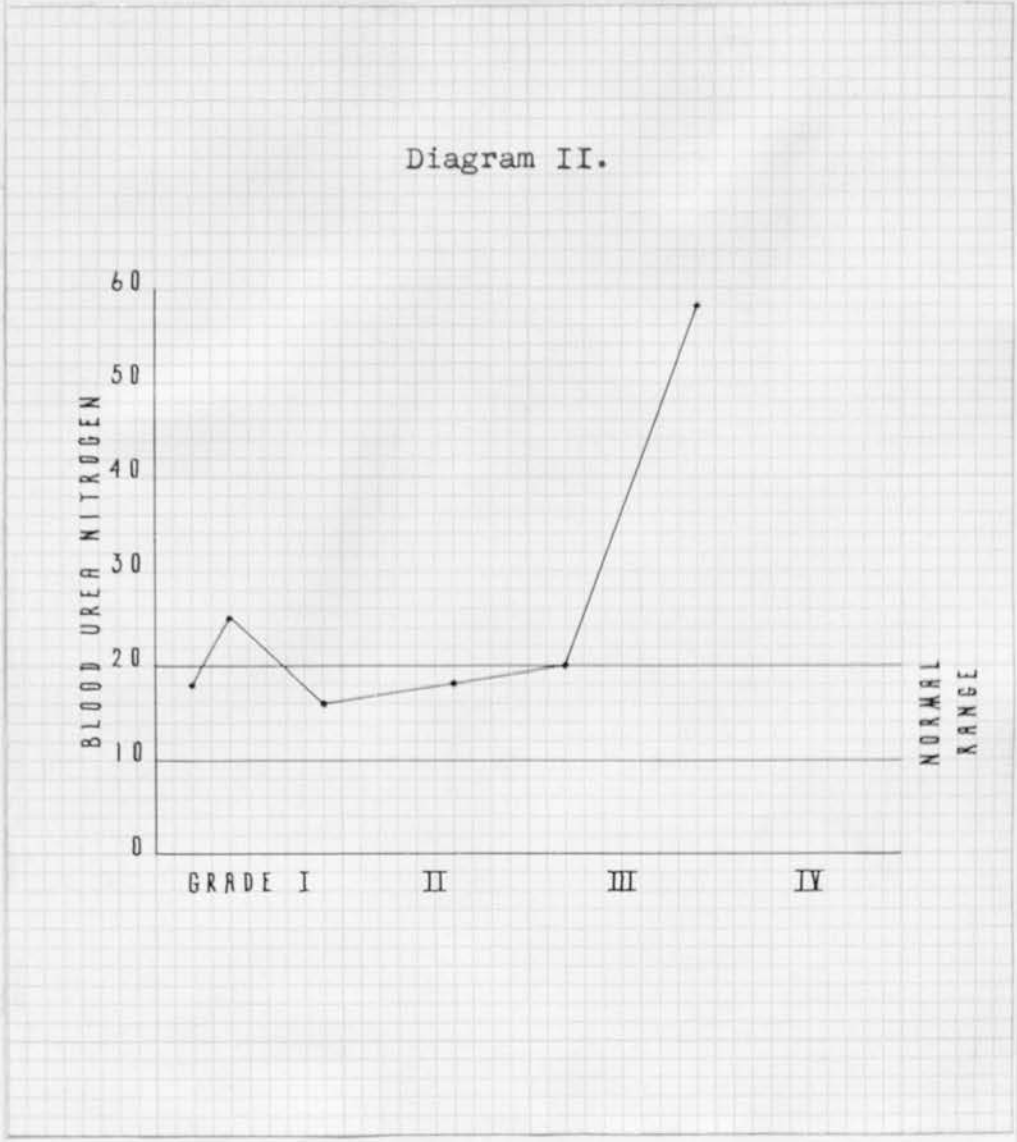


There is a definite relationship between the blood pressure levels and the fundal changes in the patients with frontal headache.

2. Fundal changes and blood urea Nitrogen.

Diagram II. illustrates the relationship between the fundal changes and the levels of the blood urea Nitrogen.

Diagram II.

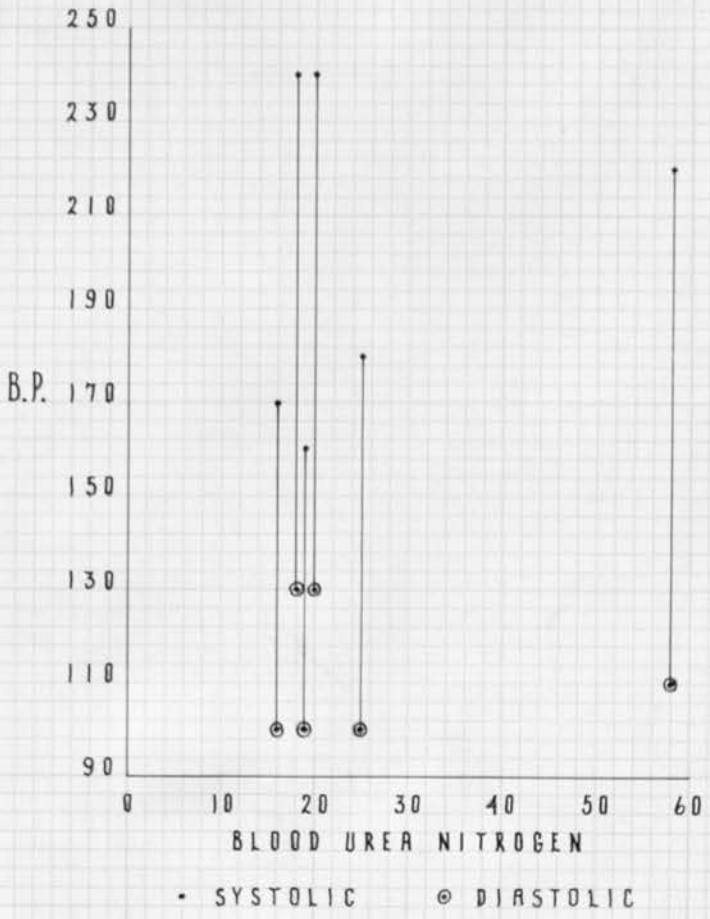


It can be seen that there is correlation between the fundal changes and the blood urea nitrogen. The higher the blood urea Nitrogen, the more severe the fundal changes.

3. B.P. levels and blood urea Nitrogen.

Diagram III. shows the blood pressure levels with the corresponding levels of blood urea Nitrogen.

Diagram III.

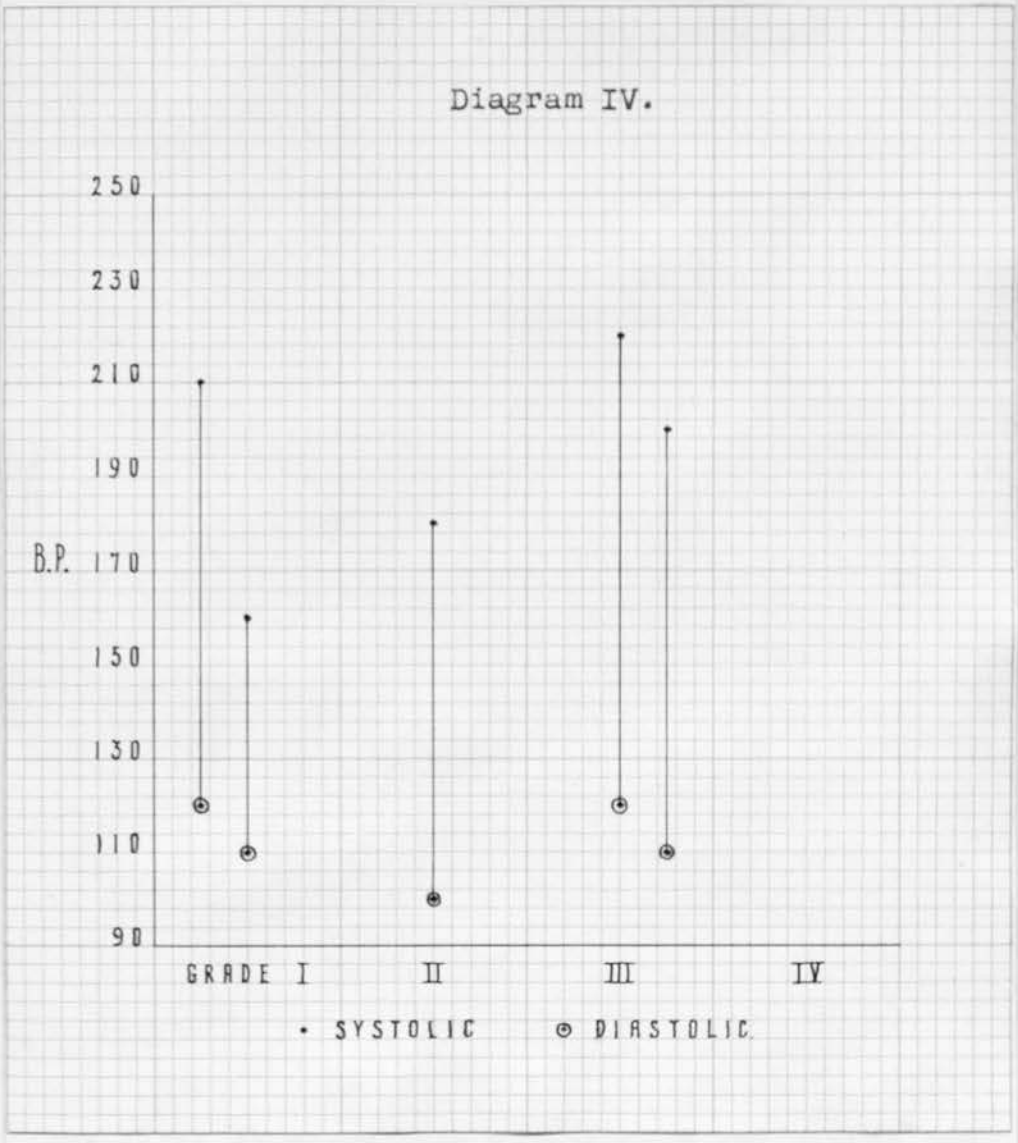


It is seen that there is no correlation between the blood urea nitrogen and the blood pressure levels.

B. OCCIPITAL HEADACHE.

1. B.P. levels and fundal changes.

Diagram IV. shows the levels of blood pressure with the corresponding fundal changes.

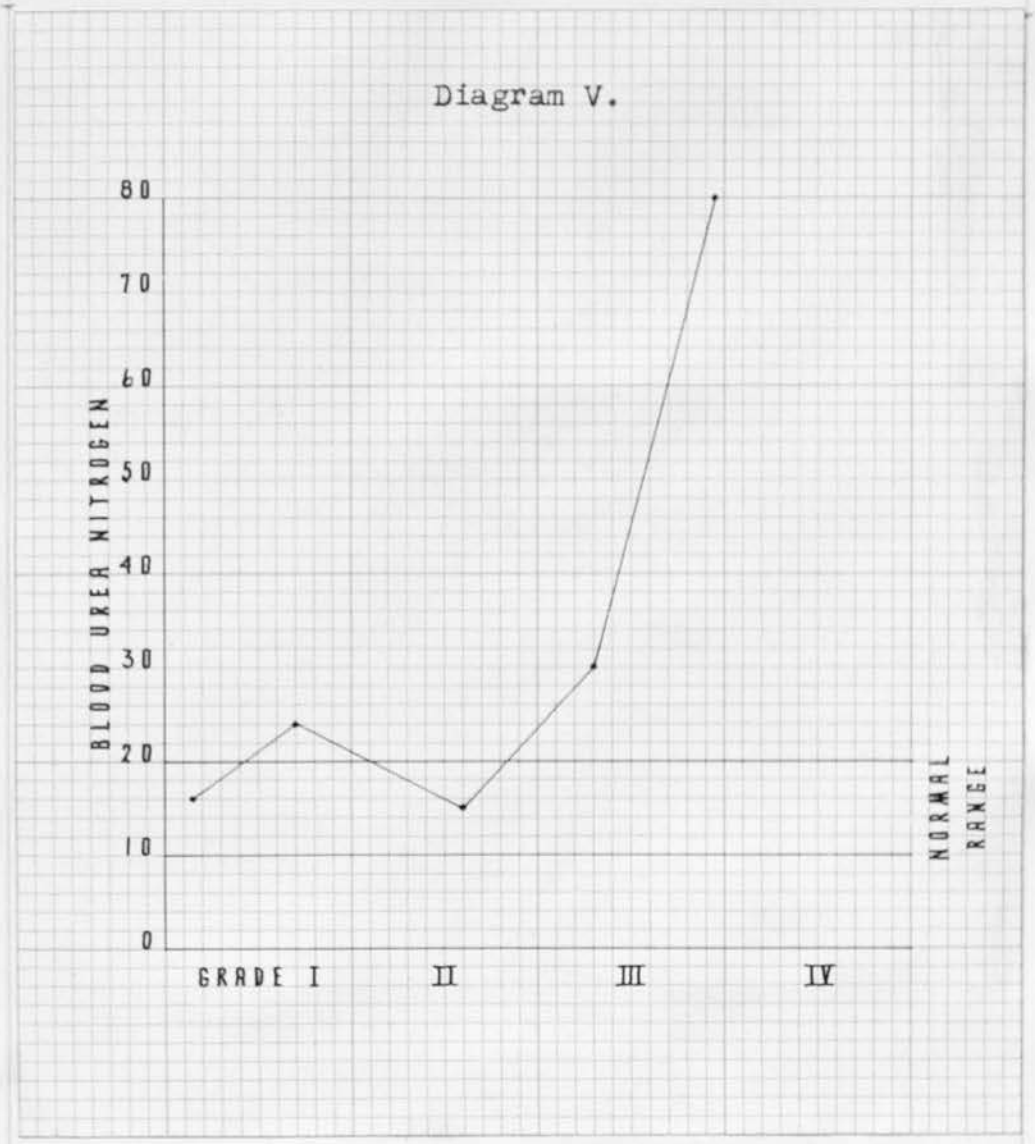


It can be seen that there is no correlation between blood pressure levels and the fundal changes.

2. Fundal changes and blood urea Nitrogen.

Diagram V. illustrates the relationship of the fundal changes and the levels of blood urea Nitrogen.

Diagram V.

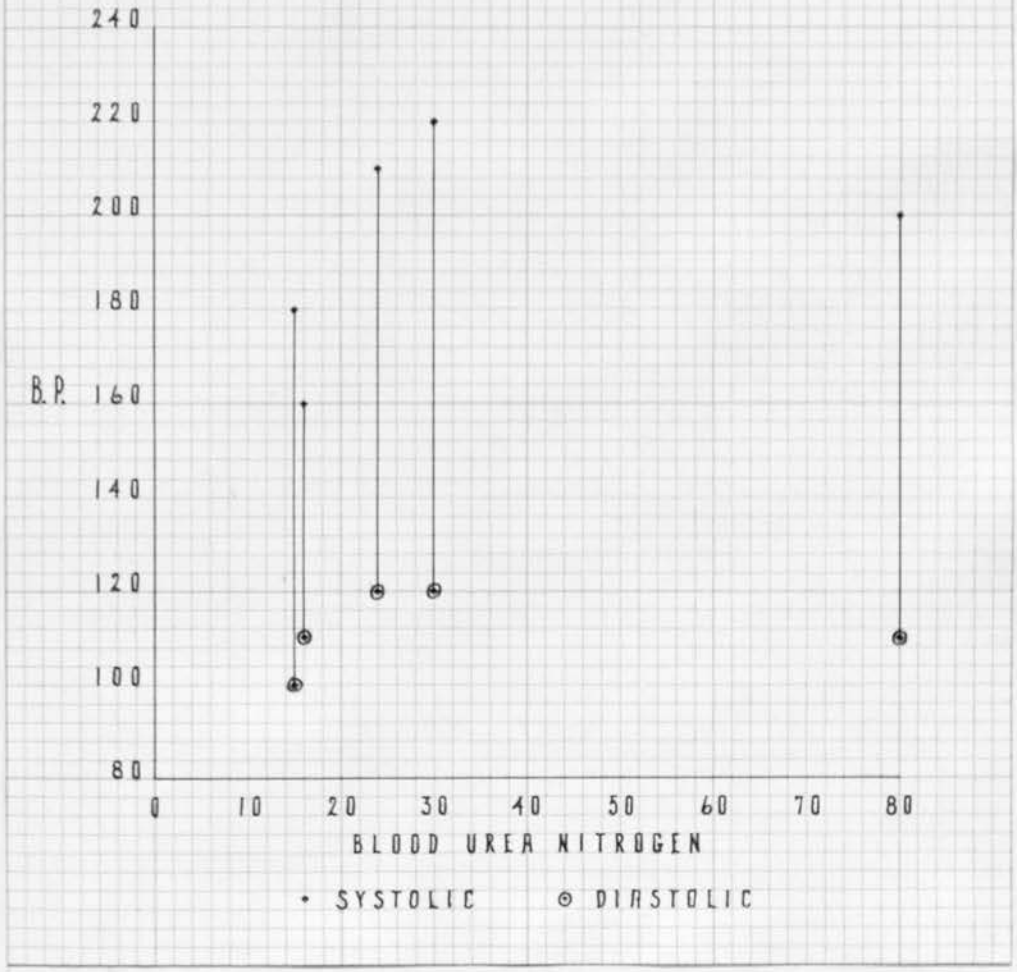


There is a correlation between fundal changes and the blood urea nitrogen. The higher the level of blood urea Nitrogen in the blood, the more severe are the retinal changes.

3. Blood urea Nitrogen and B.P. levels.

Diagram VI. shows the relationship between the height of the blood pressure and the level of the blood urea Nitrogen.

Diagram VI.



There is a correlation between the blood urea Nitrogen and the blood pressure levels. The higher the level of blood urea nitrogen, the higher is the blood pressure.

Preliminary Conclusions of the relationship between the pathological changes in patients with frontal and occipital headache.

(Diagrams 1-6).

A. 1. FRONTAL HEADACHE.

(1) Blood-pressure levels and fundal changes:

There is a definite relationship between the blood-pressure levels and the fundal changes in the patients with frontal headache in essential hypertension. The higher the blood-pressure, the more severe are the fundal changes.

(2) Fundal changes and Blood Urea Nitrogen:

There is correlation between the fundal changes and the blood urea nitrogen. The higher the blood urea nitrogen, the more severe the fundal changes.

(3) Blood-pressure levels and Blood Urea Nitrogen:

It is seen that there is no correlation between the blood urea nitrogen and the blood-pressure levels.

B. OCCIPITAL HEADACHE:

(1) Blood-pressure levels and fundal changes.

It can be seen that there is no correlation between blood-pressure levels and the fundal changes in patients with occipital headache.

(2) Fundal Changes and Blood Urea Nitrogen :

There is a correlation between fundal changes and the blood urea nitrogen. The higher the level of urea nitrogen in the blood, the more severe are the retinal changes.

(3) Blood Urea Nitrogen and B.P. levels :

There is a correlation between the blood urea nitrogen and the blood-pressure levels. The higher the level of the blood urea nitrogen, the higher the blood pressure.

The Relationship between the pathological changes in both groups are compared in Table IV.

Table IV.

	B.P. and Fundi	B.P. and Blood Urea Nitrogen	Fundi and Blood Urea Nitrogen
Frontal Headache	+	-	+
Occipital "	-	+	+

Table IV. would indicate that frontal headache in essential hypertension depends on the level of the blood pressure, which varies directly with fundal changes, and although the blood urea nitrogen and the fundal changes are correlated, there is no relationship between the blood pressure and the blood urea nitrogen.

Occipital headache however, would seem to depend more on the level of the blood urea nitrogen.

Relationship of the Severity of the Headache with Pathological Changes.

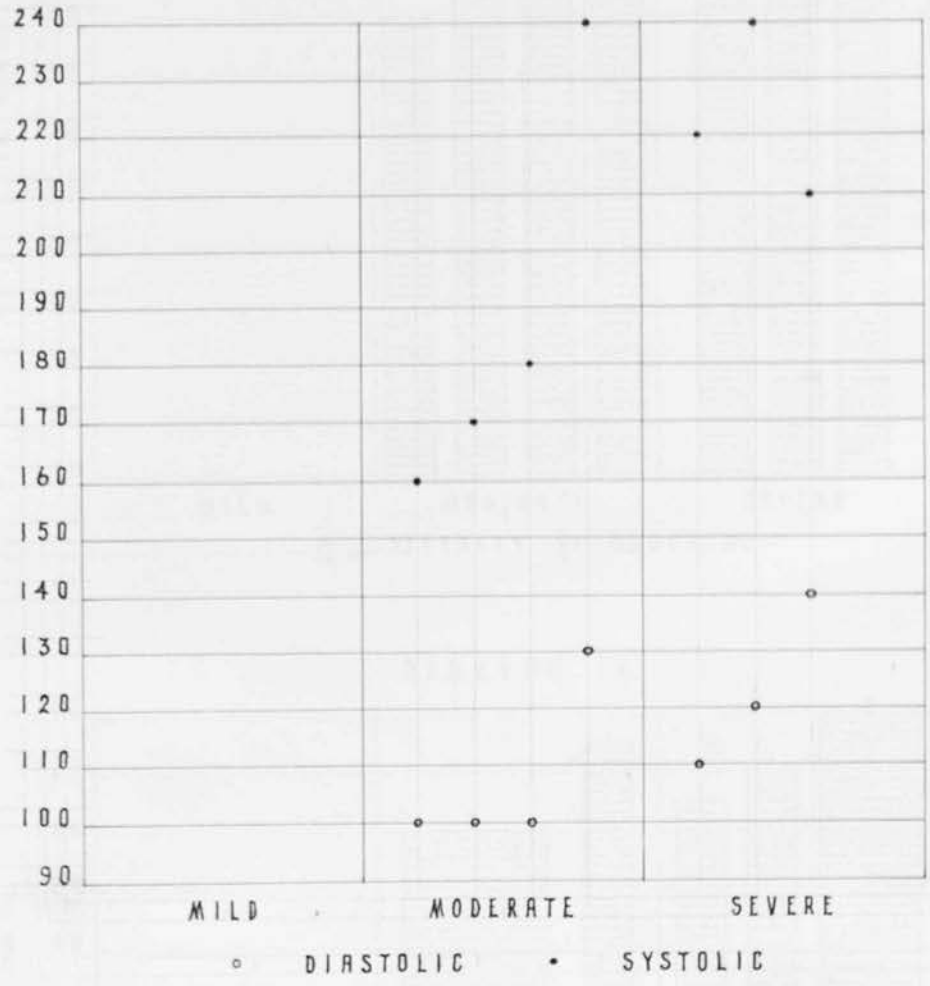
As before, the headache is divided into frontal and occipital.

A. FRONTAL HEADACHE.

1. Severity of Frontal Headache and Blood pressure levels.

Diagram VII. shows the relationship between the severity of the headache and the levels of blood pressure.

DIAGRAM VII



The range of Blood pressure can be seen in the following Table:

Table V.

Severity of Headache	The Range of Systolic Pressure.	The Range of Diastolic Pressure.
Moderate	160 - 240	100 - 130
Severe	210 - 240	110 - 140

Relationship of the Severity of the Headache with Pathological Changes.

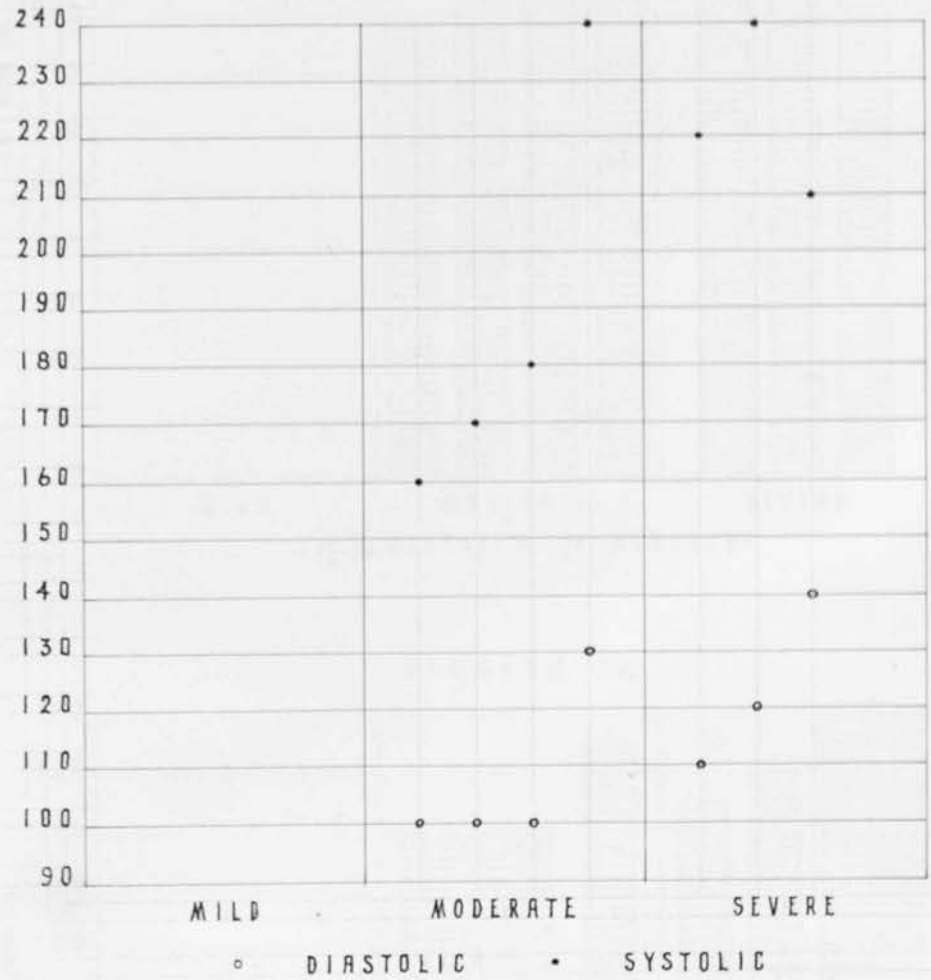
As before, the headache is divided into frontal and occipital.

A. FRONTAL HEADACHE.

1. Severity of Frontal Headache and Blood pressure levels.

Diagram VII. shows the relationship between the severity of the headache and the levels of blood pressure.

DIAGRAM VII



The range of Blood pressure can be seen in the following Table:

Table V.

Severity of Headache	The Range of Systolic Pressure.	The Range of Diastolic Pressure.
Moderate	160 - 240	100 - 130
Severe	210 - 240	110 - 140

When the severity of the headache is compared with the levels of the systolic and diastolic pressure separately as in diagram VIII. and IX., it can be definitely stated that there is a direct relationship between the severity of the headache and the levels of systolic and diastolic pressure.

DIAGRAM VIII

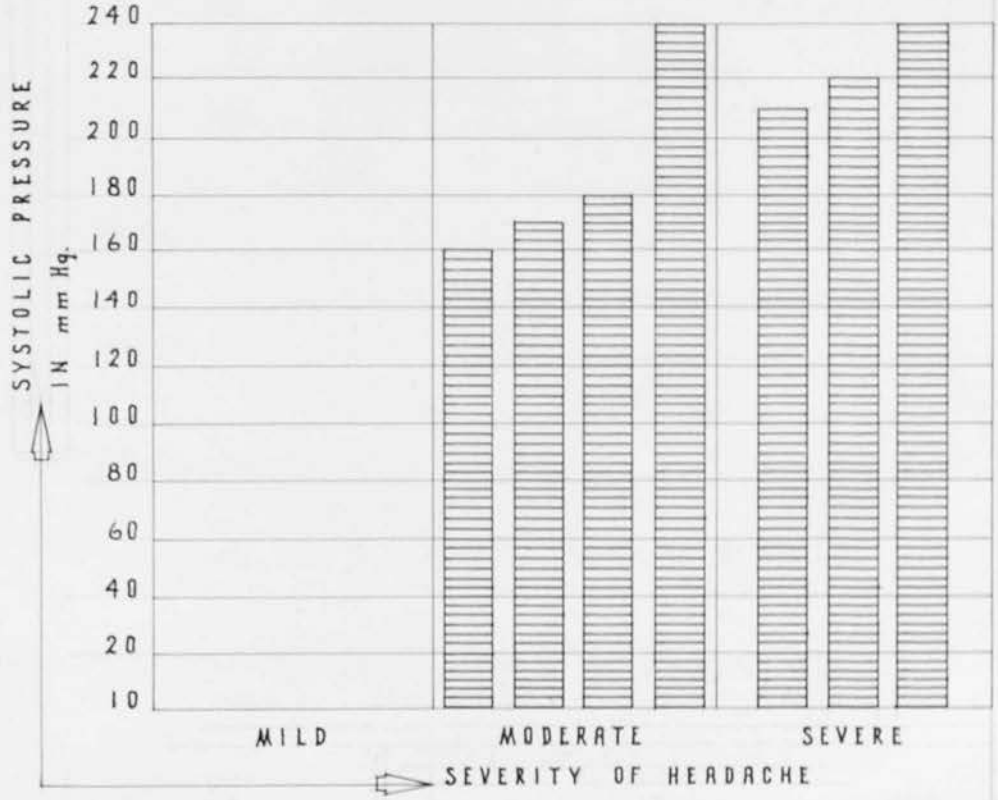
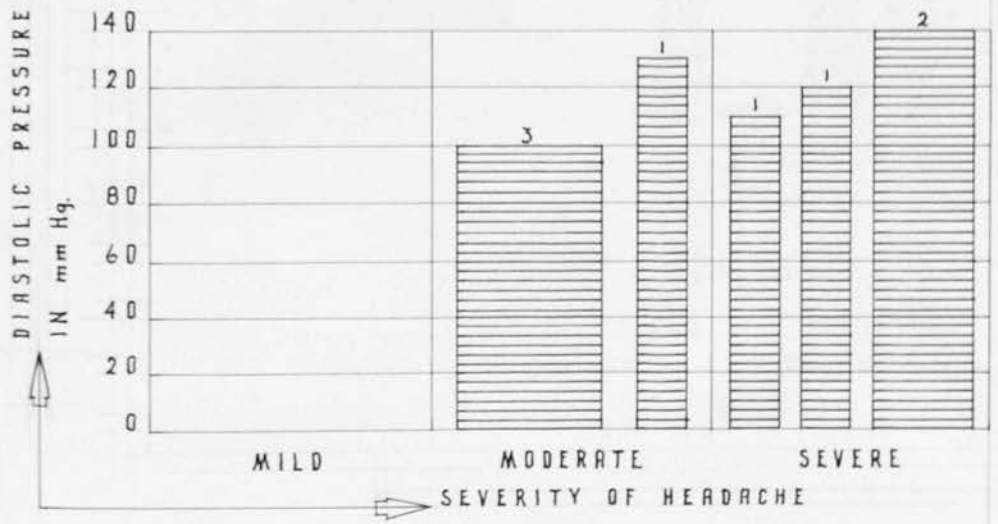


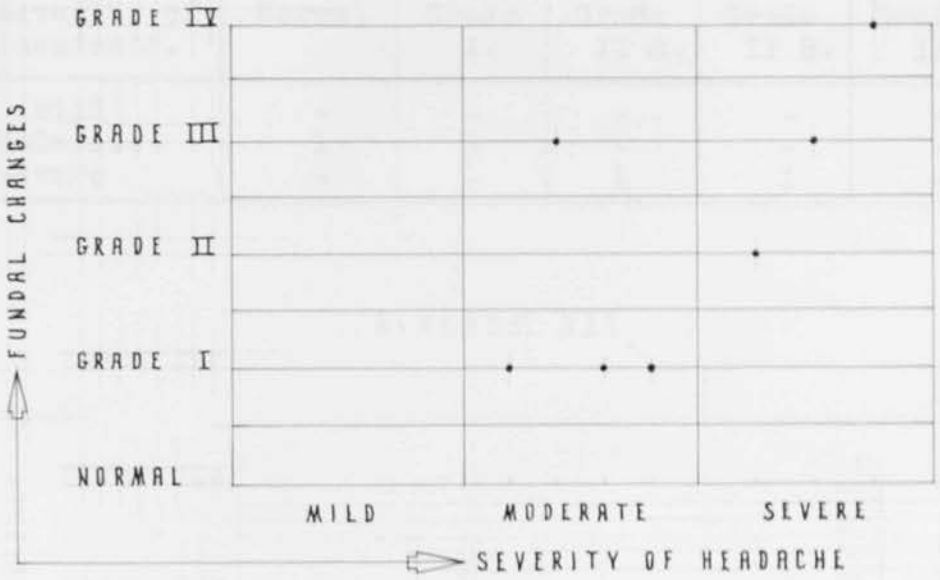
DIAGRAM IX



2. Severity of Frontal headache and Fundal changes:

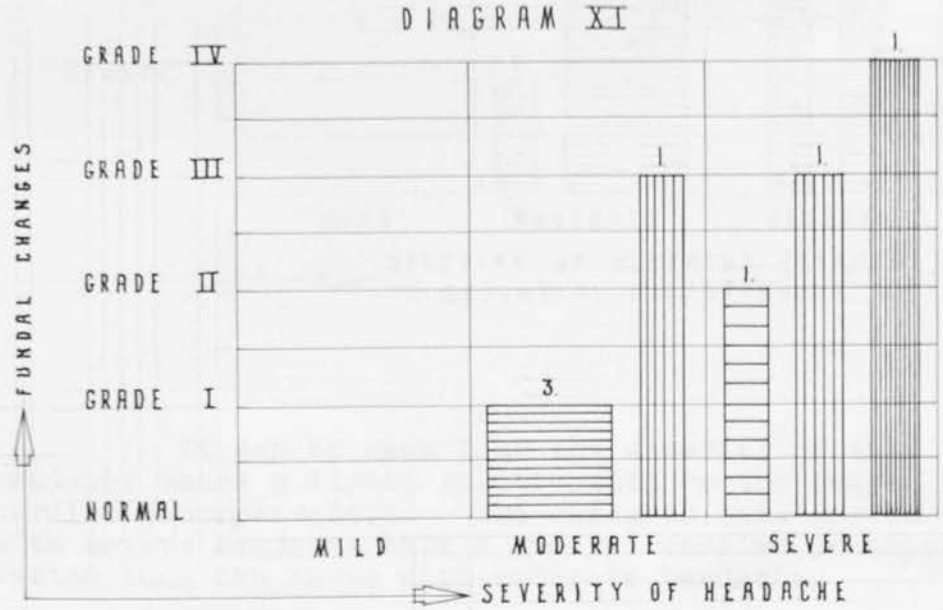
The fundal changes, graded according to the classification of Wagner and Keith have been compared with the severity of the headache in diagram X.

DIAGRAM X



This relationship is elaborated in diagram XI.

DIAGRAM XI



It can be seen that the severity of the frontal headache in essential hypertension bears a direct relationship to the fundal changes. The more severe the headache, the more severe the grade of fundal change.

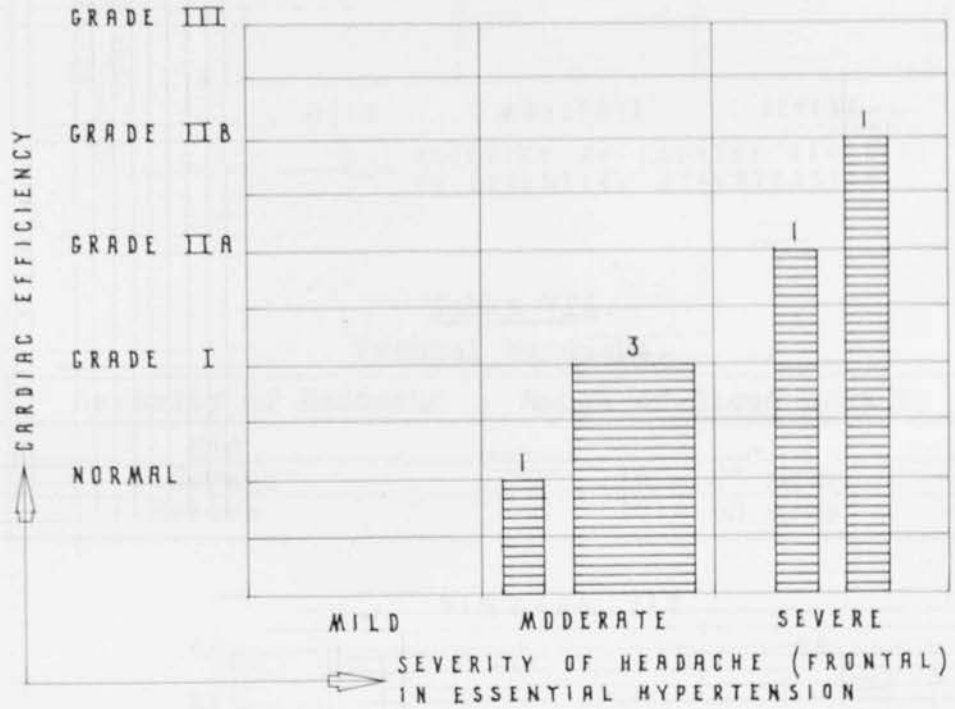
3. Severity of Frontal headache and Cardiac Efficiency.

The severity of the Frontal headache is compared with the cardiac efficiency in Table V. and also in Diagram XII.

Table VI.

Severity of headache.	Normal	Grade I.	Grade II A.	Grade II B.	Grade III.
Mild	-	-	-	-	-
Moderate	1	3	-	-	-
Severe	-	-	1	1	-

DIAGRAM XII



It can be seen that the severity of the headache bears a direct relationship to the degree of cardiac decompensation. The cases in this series with severe headache have a greater cardiac decompensation than the cases with moderate headache.

4. Severity of Frontal headache and Blood Urea Nitrogen:

The severity of the frontal headache is compared with the blood urea nitrogen in Diagram XIII.

DIAGRAM XIII

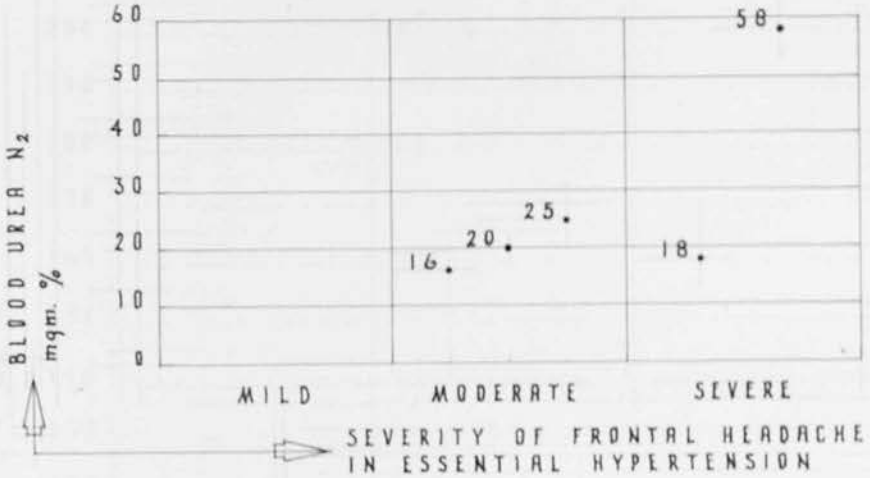
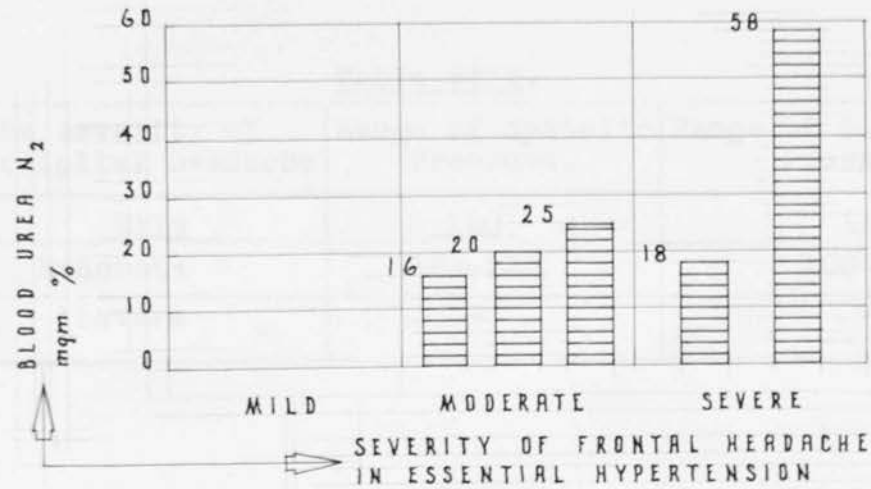


Table VII.

Frontal Headache.

Severity of Headache	Range of Blood Urea N2
Mid	-
Moderate	16 - 25 mgm%
Severe	18 - 58 mgm%

DIAGRAM XIV



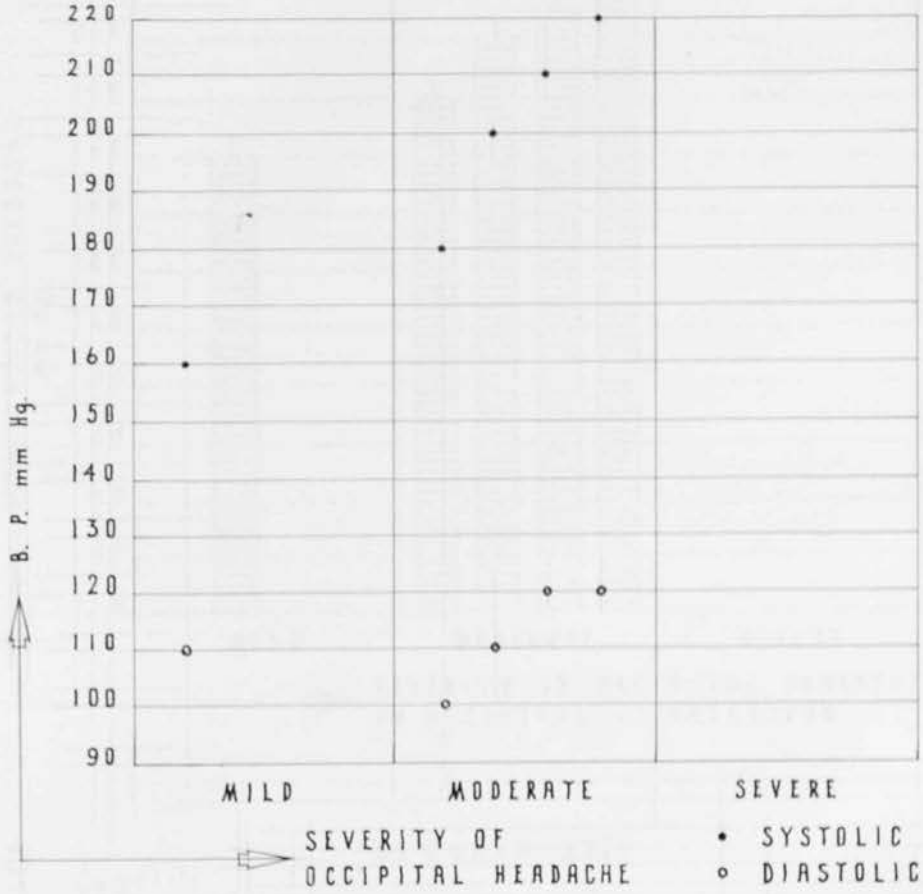
It can be seen from the above that there is only a very slight or no correlation between the severity of the frontal headache and the blood urea nitrogen.

B. OCCIPITAL HEADACHE:

1. Severity of the occipital headache and blood pressure levels.

The severity of the occipital headache is compared with the blood pressure levels in Diagram XV.

DIAGRAM XV



The range of blood pressure can be seen in the following Table.

Table VIII.

The severity of occipital headache.	Range of Systolic Pressure.	Range of Diastolic Pressure.
Mild	160	110
Moderate	180-220	100-120
Severe	-	-

The relationship between the severity of the occipital headache and the systolic and diastolic pressure are seen in Diagrams XVI. and XVII.

DIAGRAM XVI

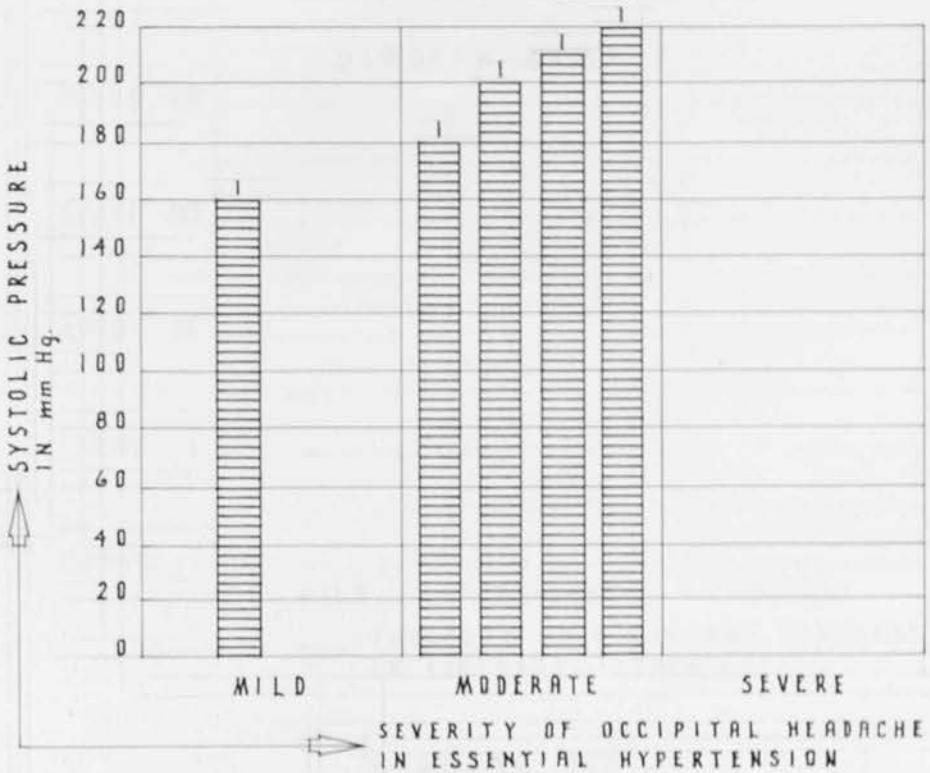
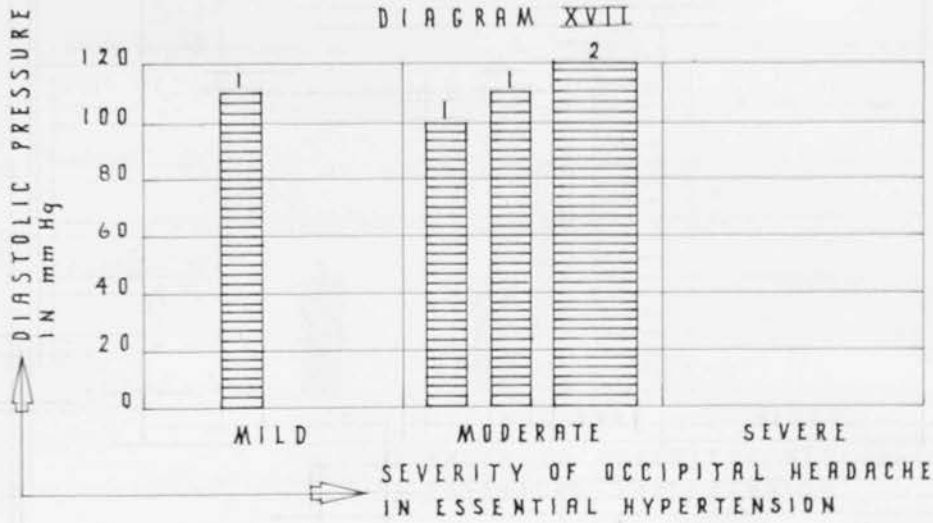


DIAGRAM XVII



These diagrams show that the severity of headache in these cases, would therefore, appear to be associated with the level of systolic pressure and not with the diastolic blood pressure.

2. Severity of the Occipital headache and fundal changes:

The severity of the occipital headache is compared with the corresponding fundal changes in Diagrams XVIII. and XIX.

DIAGRAM XVIII

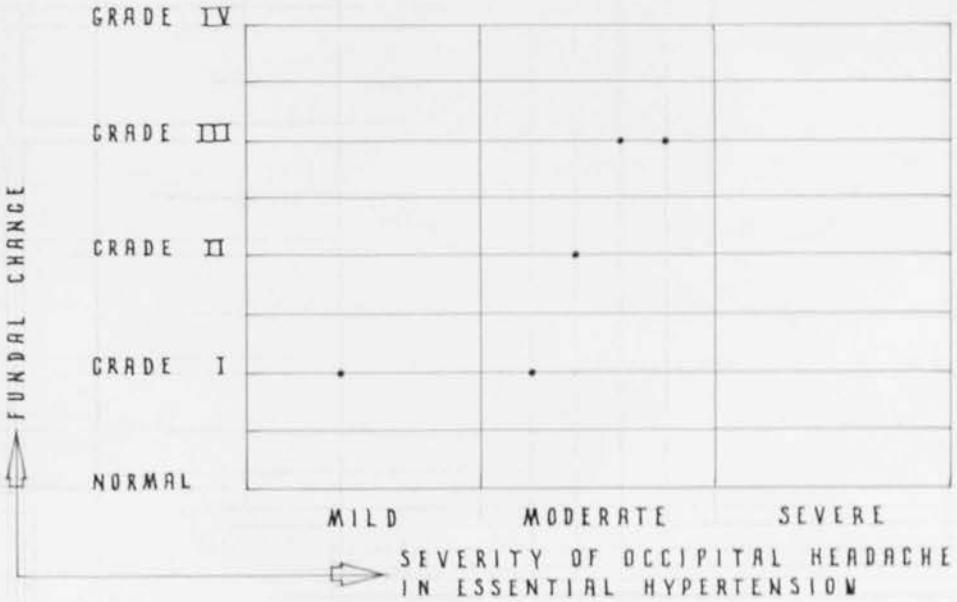
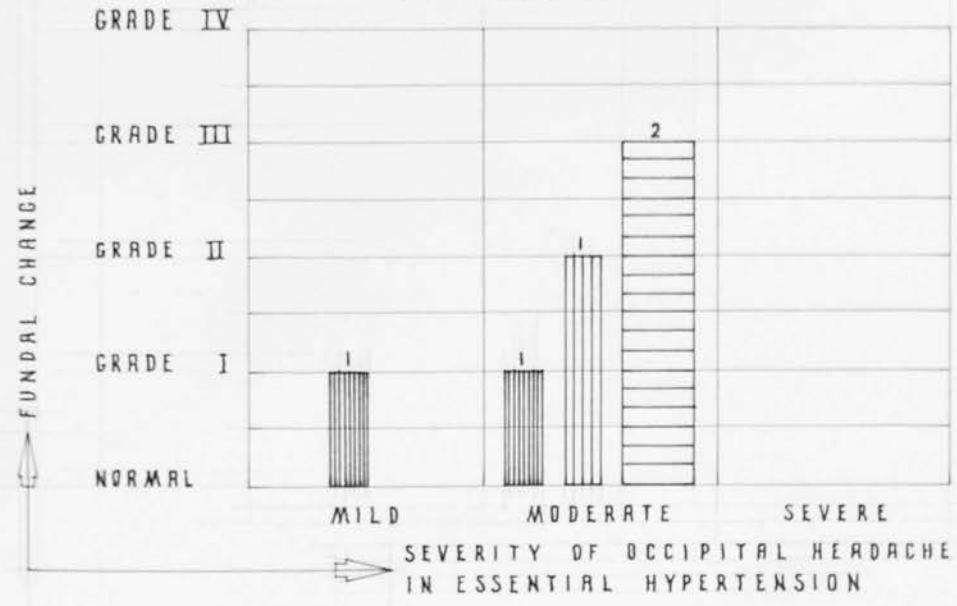


DIAGRAM XIX



These diagrams show that the severity of the occipital headache is related to the degree of fundal change. The more severe the headache, the greater the fundal change.

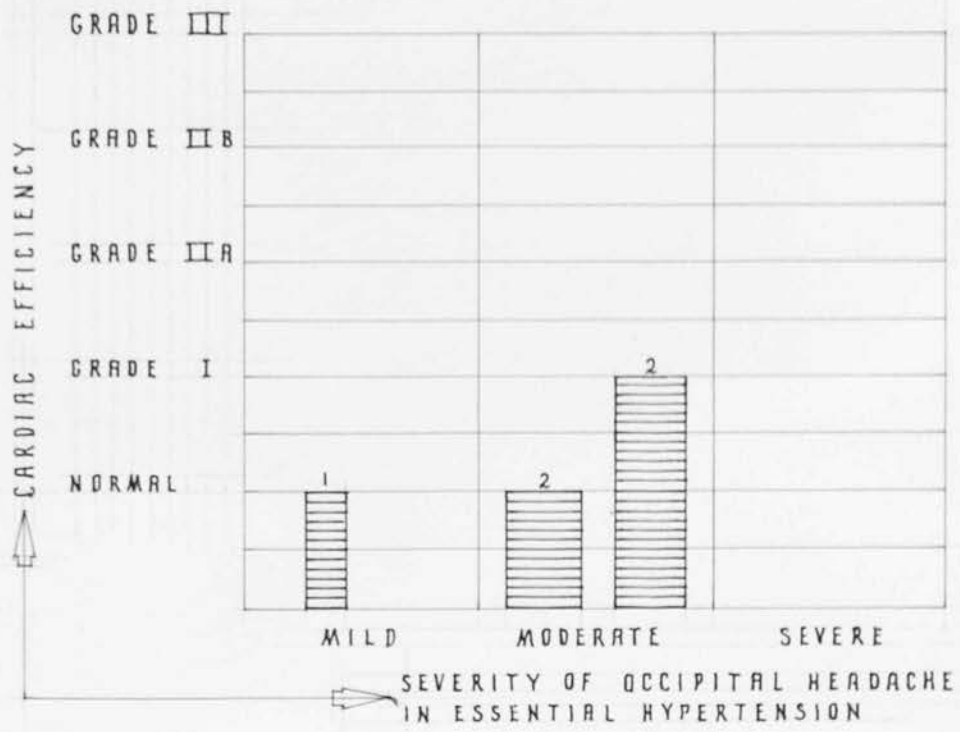
3. Severity of occipital headache and cardiac efficiency:

The severity of the occipital headache is compared with the cardiac efficiency in these cases. This is seen in Table IX., and Diagram XX.

Table IX.

Severity of headache.	Normal	Grade I.	Grade II A.	Grade II B.	Grade III.
Mild	1	-	-	-	-
Moderate	2	2	-	-	-
Severe	-	-	-	-	-

DIAGRAM XX

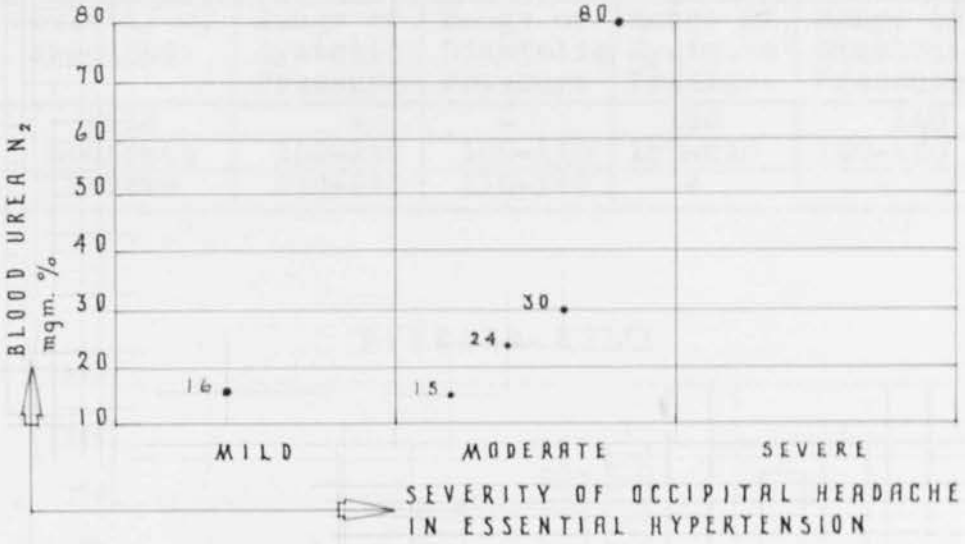


This does not show a marked relationship between the severity of the headache and the degree of cardiac decompensation.

4. Severity of occipital headache and the blood urea nitrogen.

The severity of the occipital headache is compared with the corresponding levels of the blood urea nitrogen in Diagrams XXI. and XXII.

DIAGRAM XXI



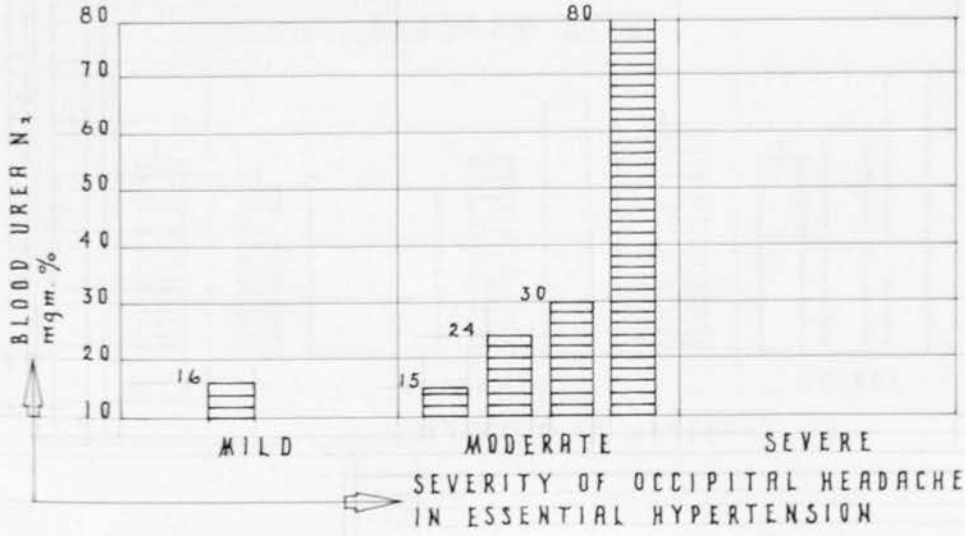
It can be seen that
(A) Mild headache: The range of the blood urea nitrogen is 16 mgm%.
(b) Moderate headache: The range of the blood urea nitrogen is 15-80 mgm%.

The range of blood urea nitrogen can be seen in the following Table:

Table X.

Severity of Headache	The range of Blood Urea N2
Mild	16 mgm%.
Moderate	15 - 80 mgm%.
Severe	-

DIAGRAM XXII



These diagrams show a direct relationship between the severity of the headache and the level of the blood urea nitrogen.

Comparison of severity of Frontal with Occipital headache and the relationship to blood pressure, Fundi, cardiac efficiency and Blood urea nitrogen.

1. Comparison of the severity of Frontal and Occipital headache with the level of blood pressure in Table X. and Diagrams XXIII. and XXIV.

Table XI.

Severity of Headache.	Range of Systolic Pressure	Range of Diastolic Pressure	Range of Systolic Pressure	Range of Diastolic Pressure
Mild	-	-	160	110
Moderate	160-240	100-130	180-220	100-120
Severe	210-240	110-140	-	-

DIAGRAM XXIII

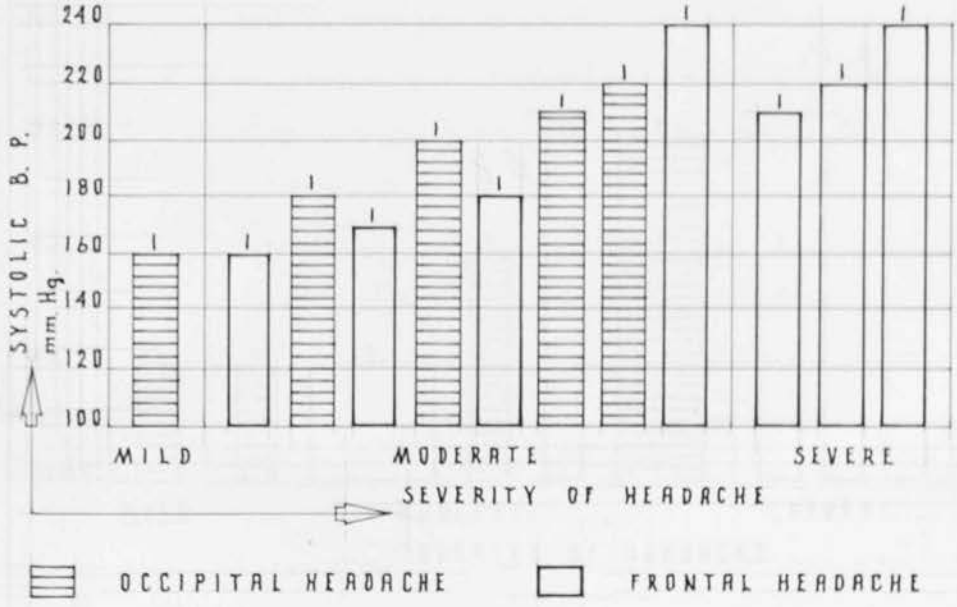
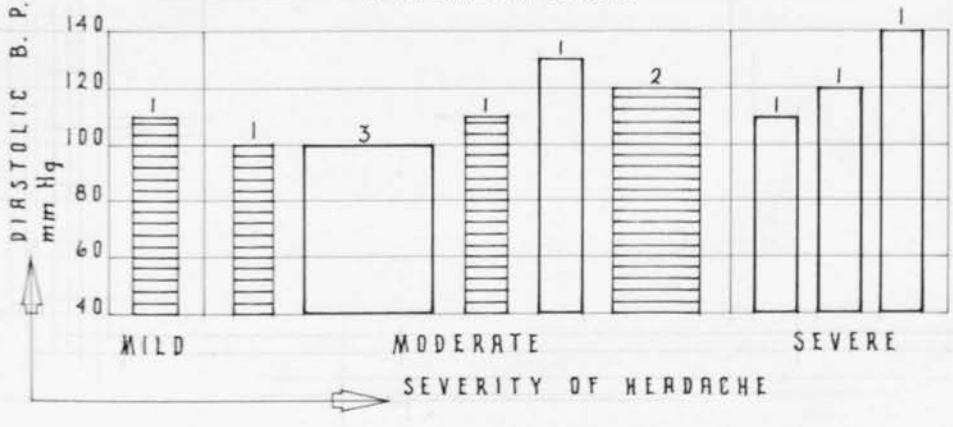


DIAGRAM XXIV

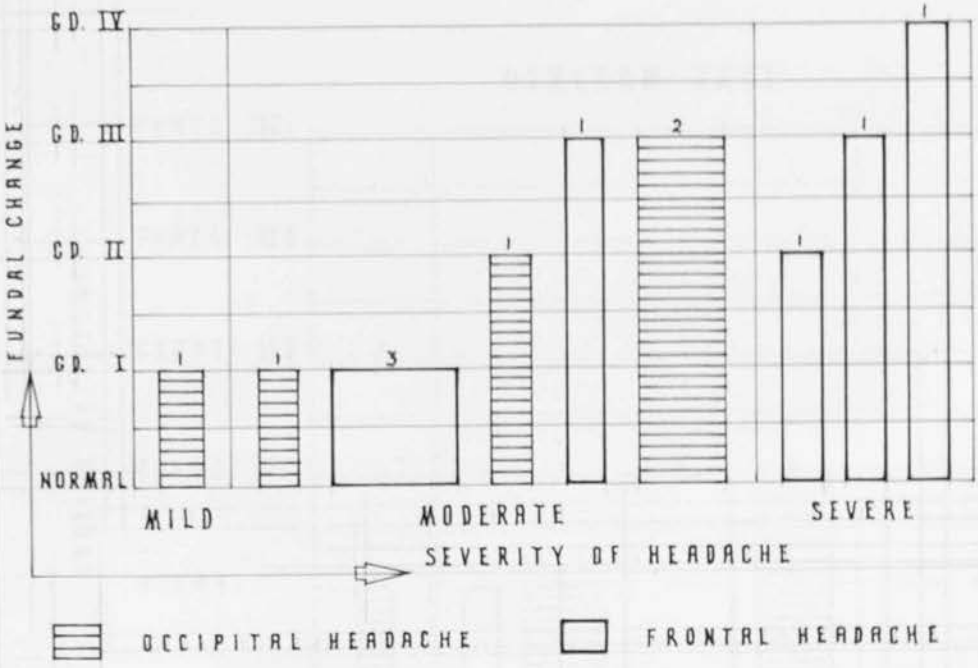


2. Comparison of the severity of Frontal and Occipital headache with the fundal changes, in Table XI. and Diagram XXV.

Table XII.

Severity of Headache	Frontal	Occipital
Mild	-	Stage I.
Moderate	Stage I-III.	Stage I-III.
Severe	Stage II-IV	-

DIAGRAM XXV

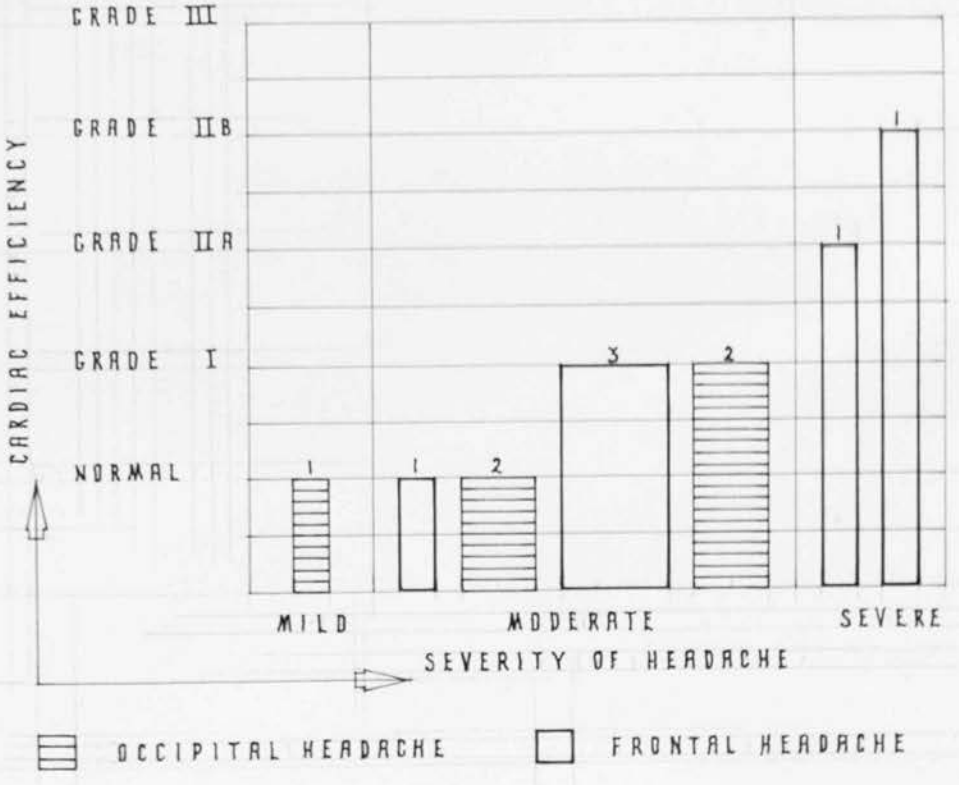


3. Comparison of Cardiac efficiency with severity of Frontal and Occipital headache in Table XII. and Diagram XXVI.

Table XIII.

Severity of Headache	Normal	Grade I.	Grade IIA.	Grade IIB.	Grade III.	Normal	Grade I.	Grade IIA.	Grade IIB.	Grade III.
Mild	-	-	-	-	-	1	-	-	-	-
Moderate	1	3	-	-	-	2	2	-	-	-
Severe	-	-	1	1	-	-	-	-	-	-

DIAGRAM XXVI

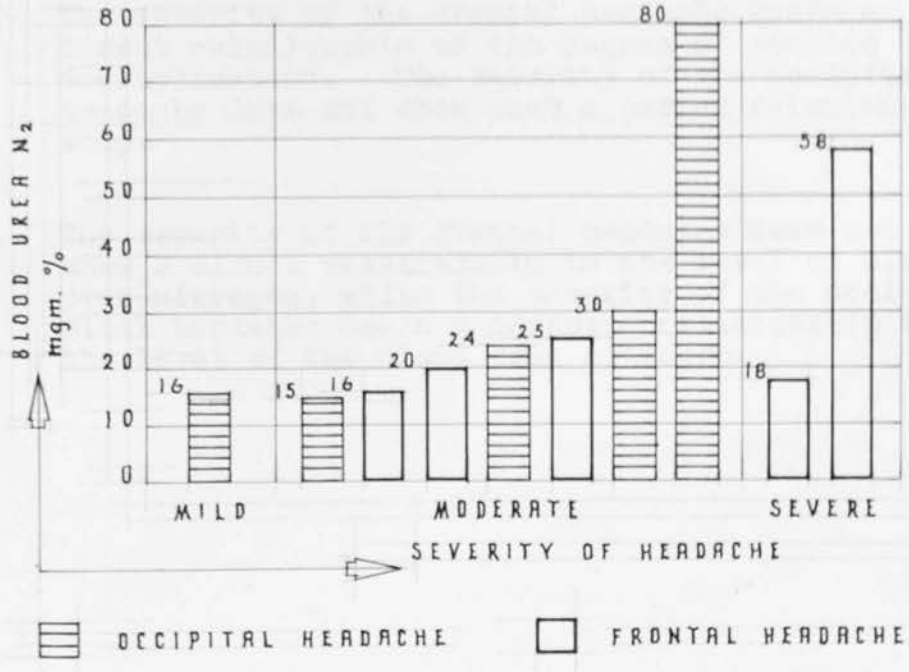


4. Comparison of Frontal headache with blood urea nitrogen and occipital headache and blood urea nitrogen in relation to its severity in Table XIII. and Diagram XXVII.

Table XIV.

Severity of Headache.	Frontal Headache Range of blood urea N_2	Occipital Headache. Range of blood urea N_2
Mild	-	16 mgm%.
Moderate	16 - 25 mgm%	15 - 80 mgm%
Severe	18 - 58 mgm%	-

DIAGRAM XXVII



Conclusions of Relationship of pathological changes with the severity of frontal and occipital headache.

1. The severity of the frontal headache bears a direct relationship with the levels of systolic and diastolic pressure.

   The severity of the occipital headache only bears a relationship to the level of the systolic pressure.

2. The severity of the frontal and occipital headache bears a direct relationship to the fundal changes. Although with headache of the same severity, the fundal changes are more marked in patients with occipital headache.

3. The severity of the frontal headache bears a direct relationship to the degree of cardiac decompensation. The severity of the occipital headache does not show such a marked relationship.

4. The severity of the frontal headache does not show a direct relationship to the level of blood urea nitrogen, while the severity of the occipital headache bears a definite relationship to the level of the blood urea nitrogen.

Discussion of the incidence of Headache in Essential Hypertension:

The analysis of the clinical findings in all these patients with headache who suffer from essential hypertension have shown that the headache is of the same character in every case but in some cases it is situated frontally and in others situated occipitally.

In essential hypertension therefore, the presence of headache, frontal or occipital is associated with several causative factors, just as the severity is associated with other diverse factors. In addition, there is a relationship between the incidence and severity of the headache with many of the pathological changes resulting from or produced by the hypertension.

Patients with frontal headache show a relationship between the levels of their blood pressure and the associated fundal changes. These fundal changes bear a direct relationship to the level of blood urea nitrogen.

There is no relationship in this series between the level of their blood pressure and the level of blood urea nitrogen.

Frontal headache in these patients is either of moderate or severe degree.

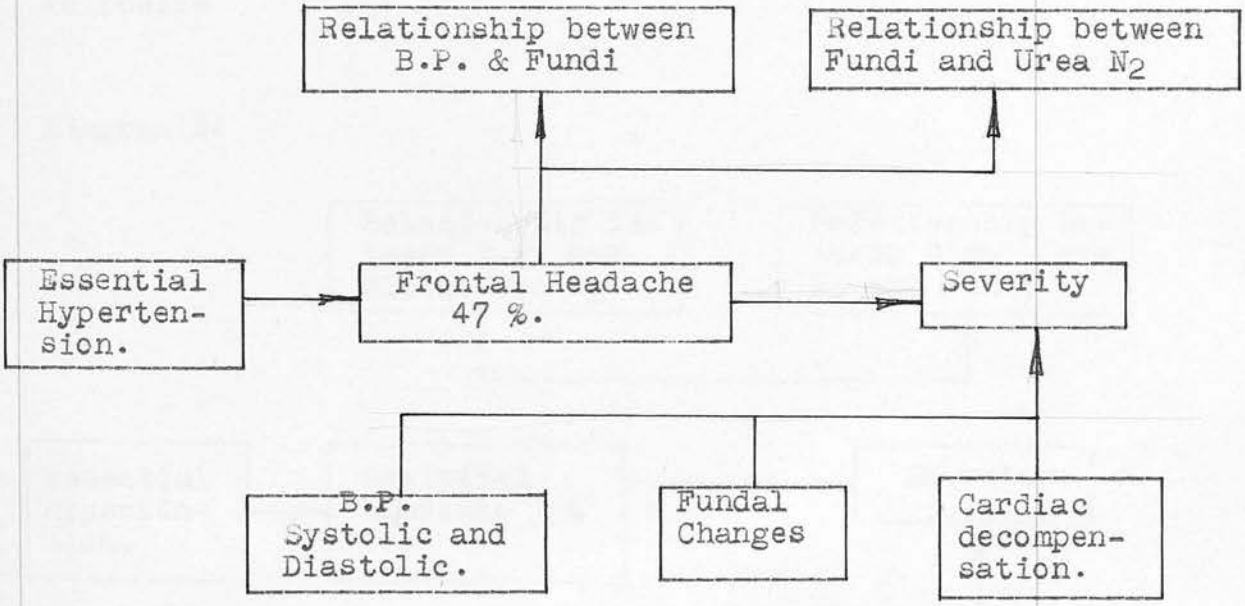
Finally, the severity of the frontal headache bears a direct relationship to :

(1) The levels of systolic and diastolic pressure.
(2) The degree of fundal changes.
(3) The degree of cardiac decompensation.

It however, bears no relationship to the level of urea nitrogen in the blood.

These observations can be represented diagramatically as follows:-

Diagram A:



In a similar way, several of the patients with essential hypertension complained of occipital headache. In these cases, there was a relationship, between the blood pressure and the blood urea nitrogen, and the fundal changes and the blood urea nitrogen. There was no relationship between the blood pressure levels and the fundal changes. The severity of the headache was not as marked in these cases and showed a direct relationship to :

(1) The level of Systolic blood pressure.
(2) The fundal change.
(3) The level of blood urea nitrogen.

There is no relationship between the severity of the headache and the degree of cardiac decompensation.

Here again, they are represented diagramatically as follows:-

Diagram.B:

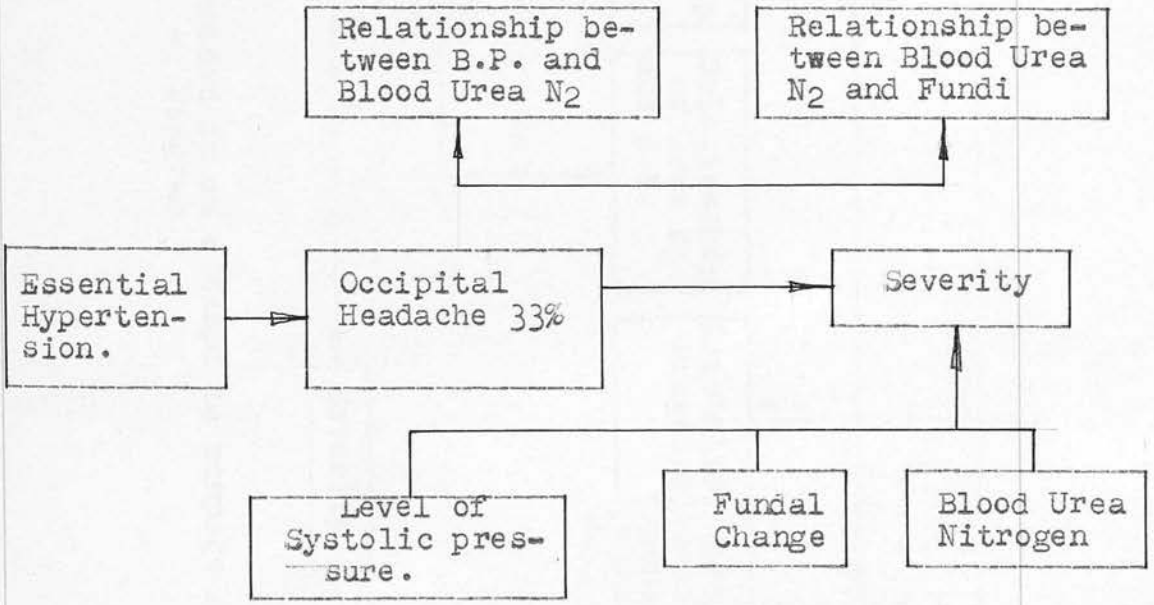
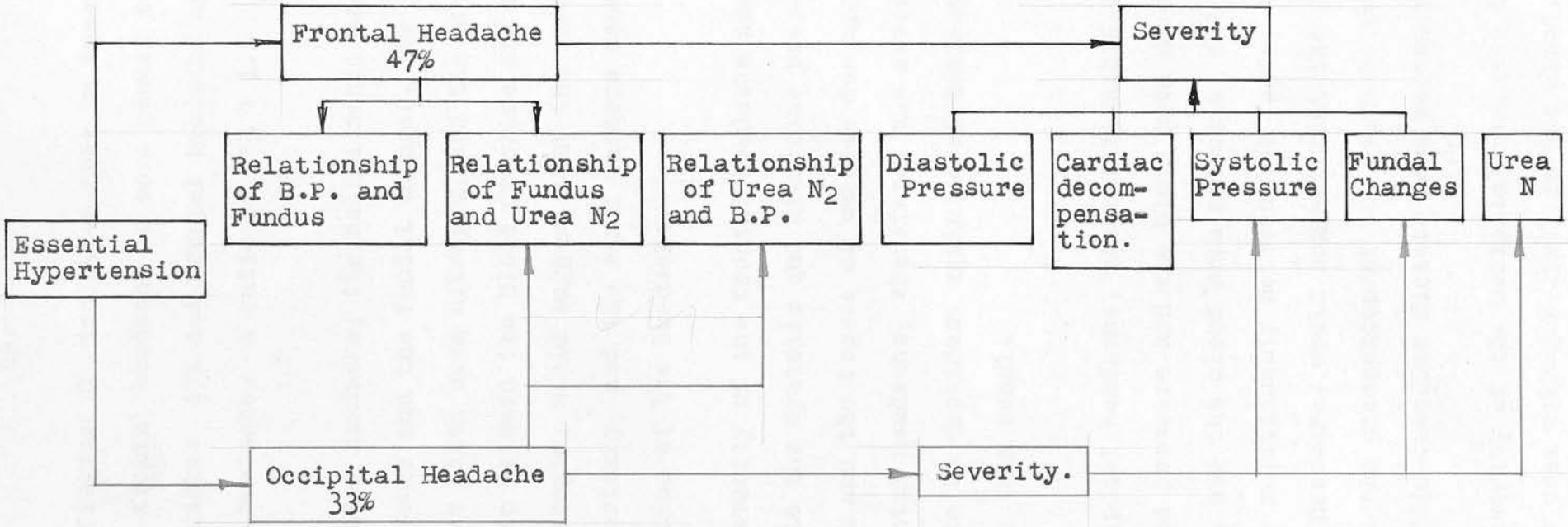


Diagram C:



These Diagrams A and B can be combined in an attempt to compare and contrast the two types of headache in essential hypertension - Diagram

The Diagram C indicates that in essential hypertension, frontal headache is more common than occipital headache: 47% had frontal headache and 33% had occipital headache. A ratio of 1·3 : 1.

In frontal headache, the relationship between the blood pressure and the fundal changes, the fundal changes and the blood urea nitrogen, and the absence of relationship between the blood pressure and the blood urea nitrogen, would suggest that the vascular factors were primary, and the renal factors secondary in the production of the headache.

The severity of the frontal headache bears a relationship to the systolic and diastolic pressure, fundal changes and the degree of cardiac decompensation.

In frontal headache, therefore, the severity would appear to be dependent again on cardiovascular changes rather than renal.

In occipital headache, the relationship between the blood pressure and the blood urea nitrogen, and the fundi, and the blood urea nitrogen, and the absence of any relationship between the fundal changes and the blood pressure, would suggest that the primary factors in the production of the headache were renal, and cardio-vascular factors were secondary.

The severity of the occipital headache is related to the urea nitrogen level in the blood,

fundal change and only to the systolic pressure. This would suggest, that the severity of the occipital headache is dependent more on renal changes rather than on cardiovascular.

Following these observations, an attempt has to be made to suggest a cause for this type of headache seen in cases of essential hypertension. When these causes are surveyed, certain precipitating factors are found in each case.

Precipitating Factors and Method of Relief :

Both groups of cases experienced headache on rising from bed in the morning.

The duration of the attack varied from half an hour to several hours in the cases of the frontal headache, and from one hour to several hours in the cases with the occipital headache.

Relief was obtained by a variety of factors:

In the case of the frontal headache:

(1) Rest and phenobarbitone gave relief in two cases.
(2) Epistaxis " " " one case.
(3) Sympathectomy " " " one "
(4) Pethidine " " " one "
(5) No relief " one "
(6) Nothing taken " one "

(headache was not severe enough to warrant anything being taken).

(7) The "head-up" position lessened the frequency of attack in 5 out of 7 cases (71%.)
(8) Intravenous hypertonic Sucrose in one of these seven cases gave relief

In the case of the occipital headache:

(1) Rest and phenobarbitone gave relief in two cases.
(2) Epistaxis and Venesection " " in one case.
(3) Aspirin " " in one case.
(4) No relief " " in one case.
(5) The "head-up" position lessened the frequency of attack in 3 out of 5 cases (60%).

These factors now will be considered in more detail.

A. Dealing first with the frontal headache:

The occurrence of the headache on rising from bed in the morning would suggest (1) sleep; and (2) Sudden movement played some part.

1. Sleep:

It would therefore be of interest to study the effects of sleep, first of all on the healthy subjects.

Kleitman (1929) has summarazied the effects of sleep as follows:

1. There is a fall in blood pressure.
2. A decrease in heart rate.
3. Respiratory rate is not changed.
4. Co_2 tension in alveolar air and arterial blood is raised due to depression of respiratory centre.
5. Basal Metabolic rate is lowered.
6. Intestinal tone is unaffected.
7. There is a smaller volume of urine secreted.
8. And thus there is a mild acidaemia.
9. Relaxation of skeletal muscle.

In essential hypertension, the systolic and the diastolic pressure is raised. The effects of elevation of this in the body will now need to be considered.

They are:-

(1) Coronary circulation:

The main aortic pressure (i.e. the mean of systolic and diastolic pressure) in the aorta is one of the main factors which control the coronary circulation (Sampson Wright). Wiggers states that there is an increased coronary blood flow in essential hypertension. This is brought about mainly by an increase in the diastolic blood flow and only slightly by an increase in the systolic blood flow. An increase therefore, in mean arterial pressure from the normal value will increase the coronary blood flow considerably. Sampson Wright has shown that in the denervated heart - lung preparation (dog) an increase in mean blood pressure from 50 to 130 mm. Hg. may increase the coronary flow from 20 to 250 c.c. per minute. When, however, Atheroma is present in the coronary arteries, this increased blood flow will be impeded and the hypertrophied myocardium will suffer from varying degrees of anoxaemia.

2. Cardiac output:

The cardiac output when the heart is not in failure, is normal in essential hypertension (Sampson Wright). The increased coronary blood flow, being adequate therefore, for the increased mass of the myocardium.

3. Venous and capillary pressure:

These two pressures are normal in essential hypertension. (Sampson Wright).

4. Arterioles:

It is known that there is arteriolar constriction in Hypertension. Many workers believe that this is the cause primarily of the elevated blood pressure; for the blood pressure is dependent on the cardiac output and the peripheral resistance. As the cardiac output is normal, and as the blood pressure is elevated, then there must be generalized arteriolar constriction. This arteriolar constriction, whether it is the cause or the result of hypertension may be generalized or occur in some organs to a greater degree than in others. If this vaso-constriction differs in degree in different regions then the blood flow will vary in these regions and the organs where there is the greatest constriction will have a greatly diminished blood flow. Measurements show that the blood flow in hypertension to the skin and brain is unchanged, (Sampson Wright) indicating that the cutaneous and cerebral vessels participate in the vaso-constriction process. Little is known about the state of the vessels elsewhere except for the kidney which has to be studied in more detail.

5. Renal blood-flow:

The renal blood flow may vary with the degree of constriction. This blood flow may be from the normal 1200 c.c. per minute to 100 c.c. (Sampson Wright). The glomerular filtration volume is much less reduced proportionally, so that, the fraction of the plasma filtered is increased. This change indicates increased glomerular pressure, and, as it is also associated with renal ischaemia, then it indicates a greater constriction of the efferent than afferent arterioles. There is therefore, a variable amount of damage to the tubules as shown by the decreased power of the tubules to excrete diodrast.

6. Blood Vessels :

All cases of hypertension show "elastosis" (elastic hyperplasia), (Samson- Wright). In malignant phase of essential hypertension, there is also arteriolar necrosis. In hypertension, therefore, the initial change is arteriolar constriction. Subsequently, the elevated blood pressure causes permanent changes. It is generally accepted that these constrictive changes are brought about by nervous and humoral factors.

7. Co_2 content of blood and the blood pressure:

By means of perfusion experiments, physiologists (Samson- Wright) have shown that a raised Co_2 tension relaxes arterioles and a lowered Co_2 tension constricts arterioles.

In addition, the vaso-motor centre can only function effectively in the presence of an adequate Co_2 tension. If there is excessive ventilation, the Co_2 tension in the alveolar air and blood fall, there is a fall in the blood pressure due to inhibition of the vaso-motor centre resulting in dilatation of the vessels in the splanchnic area (Dale and Evans).

It is desirable at this point to consider the changes in the body during sleep in hypertensive patients.

During sleep, there is a fall in blood pressure in hypertensive patients. The fall may be great or small. In these cases, in our series, the lability of the blood pressure was not great, but, nevertheless, a fall in blood pressure did occur during sleep (fall from average $\frac{210}{112}$ to $\frac{194}{110}$ mm. Hg., i.e. 16 mm. Hg. systolic and 2 mm Hg. diastolic).

The coronary circulation in hypertension is increased (Wiggers), and as mentioned before, is dependent on the level of the blood pressure. In diseased arteries and especially during sleep when the blood pressure falls, the coronary circulation is reduced). Thus there is a greater tendency to myocardial ischaemia than when awake.

In health, sleep causes no reduction in minute volume of the cardiac output below the basal level (Best and Taylor).

In hypertensive patients, however, with the fall in blood pressure, resulting in diminution of the coronary flow, the cardiac output must of necessity fall. In seven patients out of twelve with headache, the cardiac efficiency was reduced; this would be even further reduced during sleep. In this series, four out of the seven patients with frontal headache, had cardiac asthma.

The depression of the respiratory centre in health, results in lowering of the Co_2 content in the alveolar air and increase in the arterial blood. This must occur in hypertensive patients just as in healthy subjects, but due to the disproportionate supply of blood between the coronary blood flow and the myocardium with a corresponding fall in cardiac output, the Co_2 tension should be increased even further. Any left-sided failure present,(in our series there were seven such cases) with the associated pulmonary oedema, would tend to increase the Co_2 tension still further.

When the effects of sleep are discussed, it is seen that one change in the body produced by sleep is an acidaemia. (in this thesis, the term acidaemia is used, when the Ph of the blood is lowered by whatever cause and this reduction can be determined by physical measurement e.g. Co_2 combing power).

As headache, occurring in these hypertensive patients, 58% of whom showed cardiac decompensation, was present on wakening, the natural conclusion would appear to be, that the normal effects of sleep are exaggerated or altered by the associated hypertensive failure. It was decided therefore, to estimate the reaction of the blood by means of the Co_2 combining power in cases of cardiac failure to determine whether acidaemia was present, and if so, to what degree ?

In order to assess clinically, the degree of acidosis present in cardiac failure, the Co_2 combining power was estimated in ten patients suffering from many causes of cardiac failure. Patients were chosen, in whom there was no evidence of renal disease or indeed any other obvious factors which would produce acidaemia.

When Co_2 combining powers taken at 7.a.m. and during the day at 2 p.m., the following results were obtained :

Table XV.

No.of Cases	Sex	Age	Diagnosis	Co_2 combining power of the Blood	
				7 a.m.	2 p.m.
1	Male	52	Mitral stenosis	40 Vols%	46 Vols%
2	Male	56	Mitral stenosis	40 "	44 "
3	Male	48	Aortic regurgitation.	42 "	44 "
4	Male	50	Chr. Cor. Pulmonale	40 "	42 "
5	Female	36	Chr. Cor. Pulmonale	48 "	52 "
6	Female	26	Mitral stenosis	40 "	43 "
7	Female	50	Toxic Adenoma of thyroid with congestive cardiac failure.	40 "	41 "
8	Female	50	Hypertension and Coronary Thrombosis.	40 "	42 "
9	Female	56	Hypertension	44 "	48 "
10	Male	63	Coronary Vessel disease	46 "	50 "

This (Table XV.) shows that both during waking hours and on wakening from sleep in these patients with heart failure, there is a degree of acidaemia present which is exaggerated by sleep.

For, it can be seen from these figures that cardiac failure results in acidaemia. The normal range of Co_2 combining power is 53 Vols. - 73 Vols. (Van Slyke). In this series of ten patients, the

range is 52 Vols% - 41 Vols% during the day and 48 - 40 Vols% immediately on wakening (i.e. the effect of sleep).

Control Series:

Ten patients suffering from non-cardiac and non-renal conditions were chosen at random and the Co_2 combining power estimated during the day at 2 p.m. and 7 a.m. on wakening. They gave the following results:-

Table XVI.

No.of Cases	Sex	Age	Diagnosis	Co_2 Combining power of the blood	
				7 a.m.	2 p.m.
1	Female	32	Peptic Ulcer.	68 Vols%.	70 Vols%.
2	Male	50	With resolving Pneumonia	66 "	68 "
3	Male	68	Pancreatic Carcinoma.	62 "	62 "
4	Male	38	Amoebiasis	70 "	72 "
5	Female	50	Chr.Cholicystitis	64 "	68 "
6	Male	54	Early Pernicious Anaemia	66 "	66 "
7	Male	29	Peptic Ulcer	66 "	68 "
8	Female	40	Sciatica	66 "	66 "
9	Male	30	Malaria	62 "	65 "
10	Male	32	Virus Pneumonia	62 "	65 "

The above results (Table XVI.) show that there is only a mild degree of acidaemia caused by sleep in patients with no cardiac or renal impairment. Furthermore, the range of pH. is much higher than in the group with cardiac failure. In this series of ten

cases, the range is 72 Vols% - 62 Vols%. during the day and 70 - 62 Vols% immediately on wakening, (i.e. the effect of sleep).

The results, therefore show that there is:-

(1) An acidaemia present in patients with cardiac failure.
(2) This acidaemia becomes more marked during sleep.

In a normal individual, there is a diminished secretion of urine during sleep and a corresponding mild acidaemia. In hypertension, there is an increased renal blood flow, provided the kidneys are not abnormal. During sleep, this may be maintained or reduced. But due to the cardiac decompensation, however, the renal blood flow during sleep will be reduced. This will further add to the acidaemia

Thus, in hypertensive patients, during sleep, there is:-

(1) Reduced cardiac output.
(2) Increased Co_2 tension in the blood.
(3) Acidaemia

to a much more marked degree than in health.

The effects of this reduced cardiac output, increased Co_2 tension and acidaemia in the body and especially in the brain will now be considered.

<u>The reduced cardiac output produces</u>:-

(1) A fall in systolic blood pressure.
(2) A diminution of blood flow.
(3) An increased venous pressure.

Increased Co_2 tension produced dilatation of Arterioles.

Acidaemia will act in a similar way to an increased Co_2 tension and produces further dilatation of capillaries.

It is appropriate at this point to pause a a moment to consider the effects of increased oxygen consumption, perhaps producing a relative anoxaemia and the effects of excess Co_2 production, on the body. Because during sleep and in heart failure, we know there is an excess Co_2 tension in the blood and also in patients with heart failure there is an increased oxygen consumption with a corresponding increase in B.M.R. (Sampson Wright). The effects of both these mechanisms appear to be important factors in these cases.

Oxygen and Carbon dioxide tension in Normal Individual:

In normal people, the oxygen saturation of the mixed venous blood can be obtained indirectly using the Douglas and Haldane method or directly by collecting a sample of blood from the right auricle by cardiac catheterization. Arterial blood may be obtained by direct puncture of a peripheral artery.

It is found that O_2 and Co_2 tension in arterial and mixed venous blood at rest is:(Sampson Wright)

	O_2 Tension	Co_2 tension
Mixed venous blood	40 mm (14 c.c.)	46 mm (52 c.c.)
Arterial blood	80-90 mm (19 c.c.)	40 mm (40-56 c.c.)

Cerebral blood flow in normal individual :-

(Estimates based on arterial-venous (A-V) oxygen difference).

In man, the O_2 content of the carotid artery and jugular vein may be determined. It is found that, compared with its blood supply, the human brain has a high level of metabolism - the venous blood leaving the brain is therefore, more extensively reduced than elsewhere in the resting body.

The average cerebral arterial-venous difference is 6·2 c.c. (Sampson Wright). (Arterial 02 content 19 c.c. and jugular vein 12·8 c.c. - 6·2 c.c.)

We know that the R.Q. of the brain is 1, indicating that it uses carbohydrate exclusively for combustion. (Sampson Wright).

By means of the Fick principle, or by Ferris method, knowing the above factors (Ferris), the cerebral blood flow can be determined and it is found that:-

(1) The cerebral blood flow is reduced :-

(a) By a fall in arterial pressure.
(b) Over-ventilation, lowering the arterial Co_2.
(c) By a rise in intracranial pressure.

(2) The cerebral blood flow is increased:-

(a) By a rise in arterial pressure.
(b) By Co_2 excess.

The blood flow through the brain, therefore, varies directly with the blood pressure. Because the skull is an inexpandible box then compensatory dilation of the cerebral vessels can only occur if cerebro-spinal fluid is displaced.

Dilation of cerebral vessels may also occur when there are local changes in the chemical composition of the blood e.g. Co_2 excess or O_2 lack.

Experimental work on cerebral circulation:

In experimental work on cats by the thermo-electric method of Gibbs, Norcross has studied the blood flow through the parietal area of the cat's brain.

He found that inhalation of Co_2 caused a tremendous increase in blood flow, and this increase seemed to be roughly proportional to the percentage of the gas in mixture inhaled. The blood pressure was slightly raised in most experiments. The cerebro-spinal fluid pressure was markedly increased in all experiments, and this increase was greater with gas mixture having a high percentage of Co_2.

Inhalation of pure O_2 caused the blood to decrease slowly and remain at a lower level as long as the gas was administered.

The marked effect of Co_2 has been emphasised by all recent investigators and is generally concluded that this effect is of primary importance (Norcross). The increase in the blood flow is of a magnitude which so far outstrips anything produced by nerve stimulation that Schmidt and Pierson and Wolff and Lennox postulated that the chemical constituents of the blood is a far more important factor in the regulation of cerebral blood-flow than is vaso-motor control.

The experiments of Jacobi, Schmidt and others on cerebral circulation indicate that the alteration of blood pH from whatever cause would affect the size of the minute vessel of the brain.

Wolff and Lennox have stated that the chemical composition of the blood plays an important part in the regulation of the intracranial circulation.

It can be categorically stated, therefore, from clinical observations, physiological and animal experiments that there is:-

(1) An excess of Co_2 in the blood in cardiac failure.
(2) This increased Co_2 tension results in cerebral arteriolar dilatation and increased cerebral blood flow.

2. Sudden Movement:-

In spite of the reduced cardiac output, the increased saturation of Co_2 and the acidaemia, which occur during sleep in these hypertensive patients, the

cardio-vascular dynamics of the blood circulation become stabilized during sleep. Any sudden movement, made by the patient on wakening, will disturb these changes. The cardiac output is thus suddenly increased, the Co_2 tension of the blood will just as suddenly be reduced, and the blood flow through the kidney will be increased, with a corresponding reduction in acidaemia. The headache, therefore, may be brought about by the factors that have occurred during sleep, suddenly being altered on wakening.

Physiologists (Sampson Wright and others) have demonstrated that the vessels of the brain take part in the vascular changes such as vaso-constriction which occur in hypertension. One can therefore, assume that these cerebral arterioles will respond to change in Co_2 tension in the blood as do arterioles elsewhere.

The dilatation of the cerebral arterioles brought about by the increased Co_2 tension and mild acidaemia during sleep will result in traction on the pia mater and a displacement of cerebral-spinal fluid. This however, occurs gradually. This spinal fluid is absorbed into the venous sinuses and thus into the great veins. A degree of venous engorgement which must of necessity occur in left ventricular failure due to the pulmonary hypertension, will tend to impair this drainage. During sleep, therefore, the hypertensive patients have a gradual dilatation of the

cerebral arterioles and a gradual increase in volume of cerebro-spinal fluid.

On wakening and making a sudden movement e.g. on rising from bed, the cardiac output is suddenly increased, the venous return is increased and the blood pressure suddenly elevated even to its original level. We thus have therefore :

(1) An increased cardiac output.
(2) An increased venous return.
(3) An elevation of blood pressure.

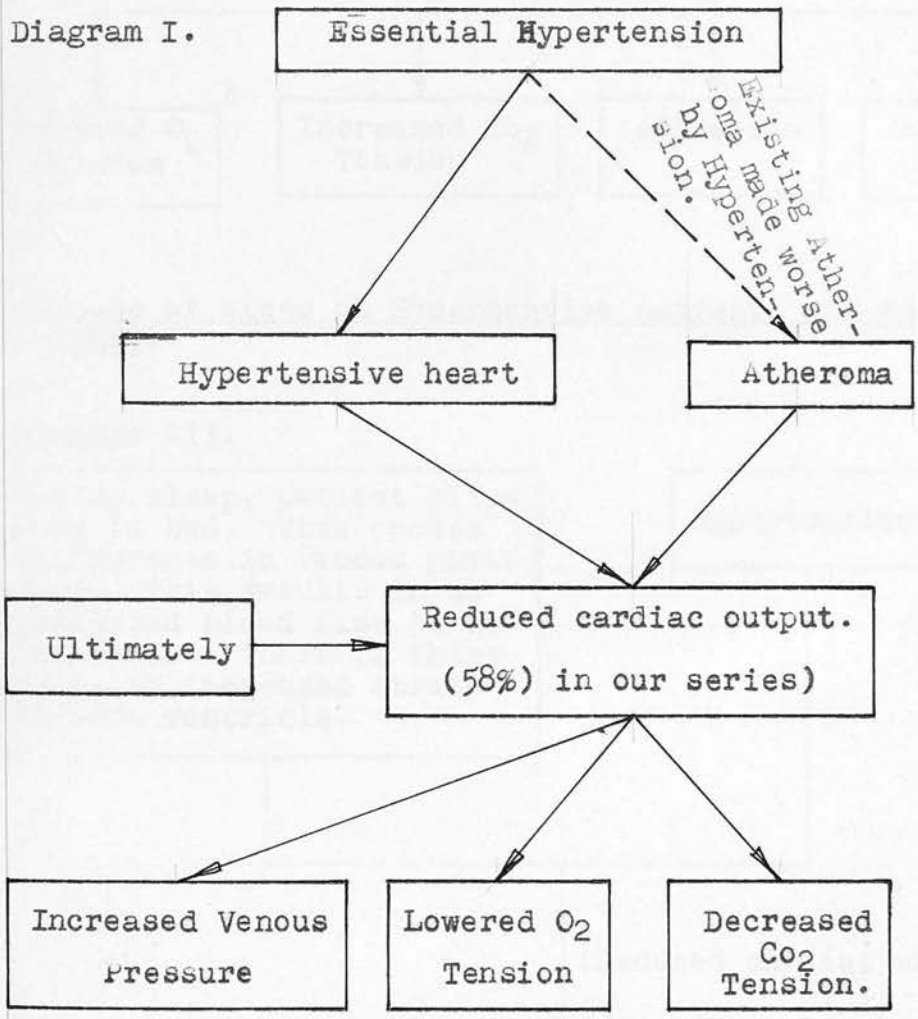
The effects of this, on the dilated cerebral arterioles will be dramatic. The cerebro-spinal fluid volume, due to the increased venous return, is rapidly restored to normal, the sudden increase in cardiac output and the sudden elevation of blood pressure will affect the already relaxed cerebral arterioles and thus distend them further to the point of producing pain. This theory receives the support of Wolff, (1943), who states that a cranial artery only slightly relaxed, for whatever reason, would not distend as much, and possibly not to the point of producing pain if the blood pressure were low. When, however, the pressure is increased, the distension is greater and therefore pain is readily produced. In other words, a degree of change in the contractile state of the arterial wall, compatible with comfort, when the blood pressure is average, is associated with pain, when the blood pressure is elevated.

Our clinical observations would suggest, therefore, that the gradual dilatation of the cerebral vessels, which occurs as a result of changes in the body during sleep in hypertensive patients is not associated with pain. When the blood pressure suddenly rises on wakening, then the dilated vessels become more distended and pain is produced. This pain is throbbing in character and lasts several minutes or several hours and is identical in character to the pain produced by histamine injection which produces a sudden dilatation of the cerebral arterioles. (Wolff (1943) and Pickering). (1933 -34 (a))

The changes can be represented by means of the following Diagrams:

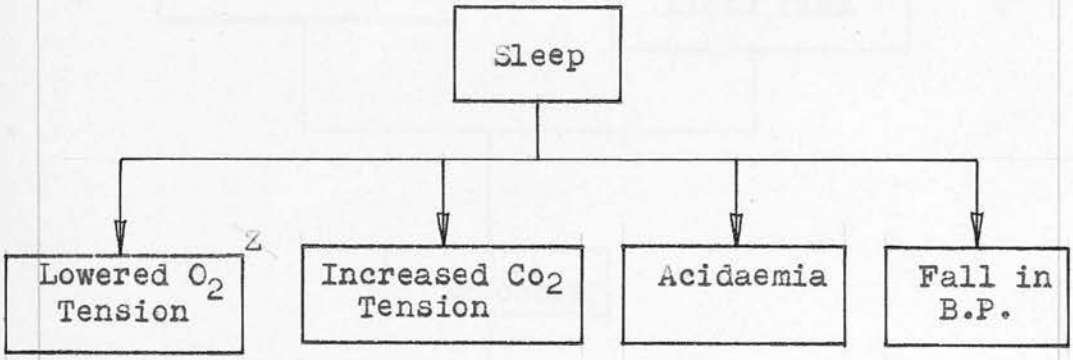
Effects of hypertension on the body.

Diagram I.



Effects of sleep on the Normal Individual (Kleitmann).

Diagram II.



Effects of sleep on Hypertensive patients are exaggerated.

Diagram III.

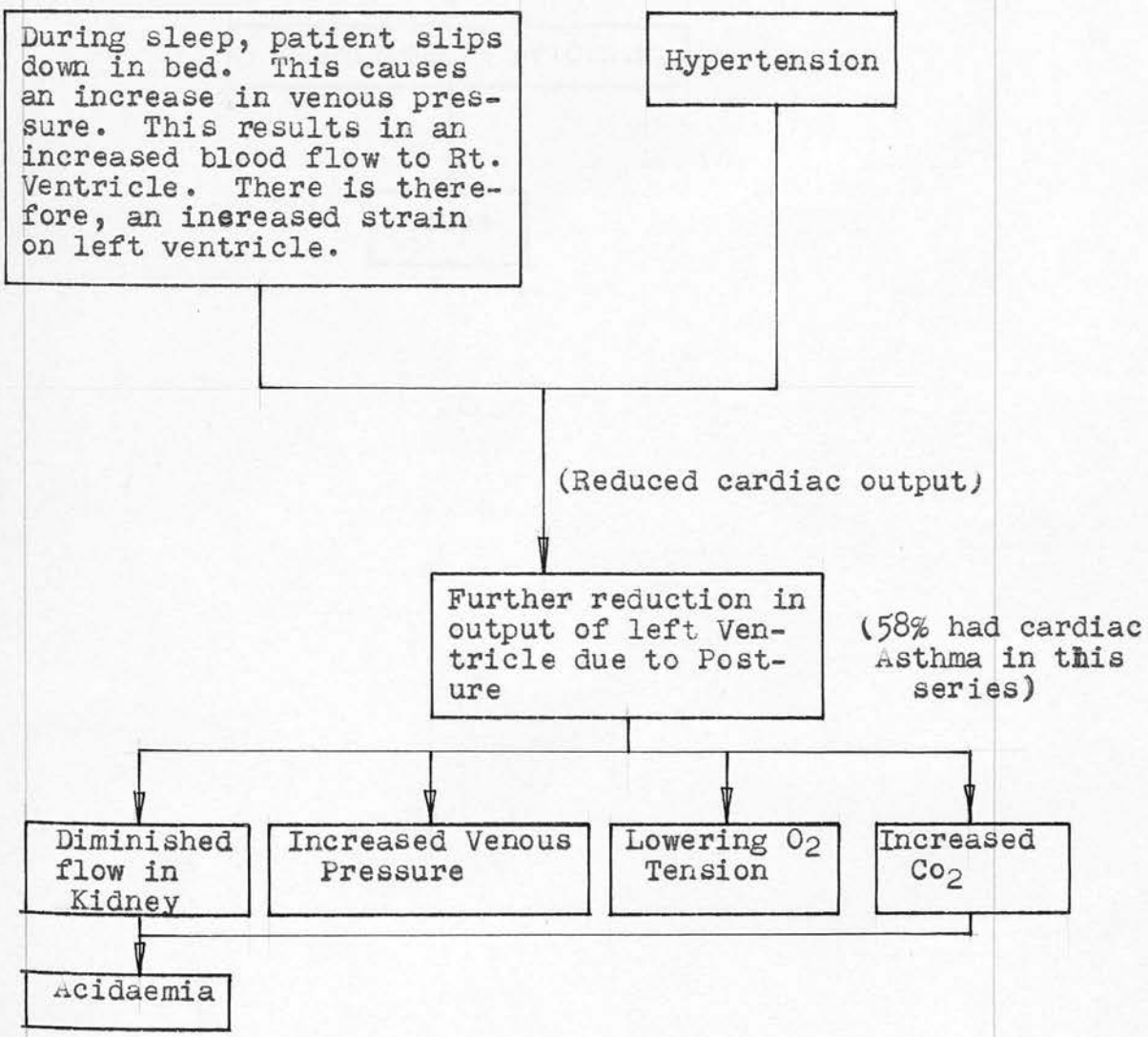
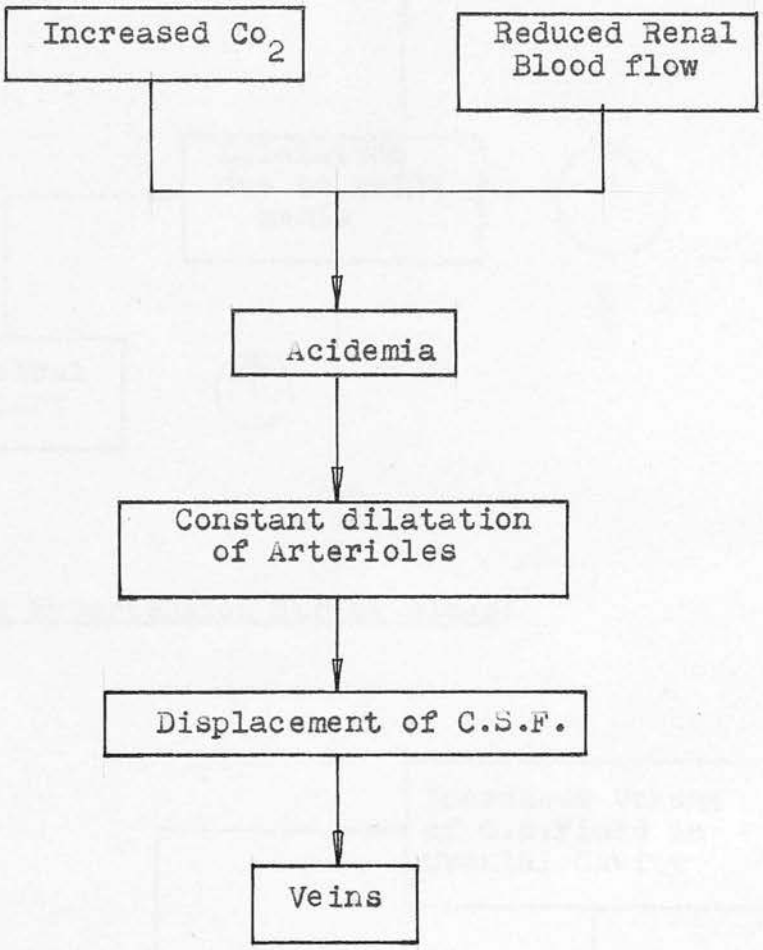
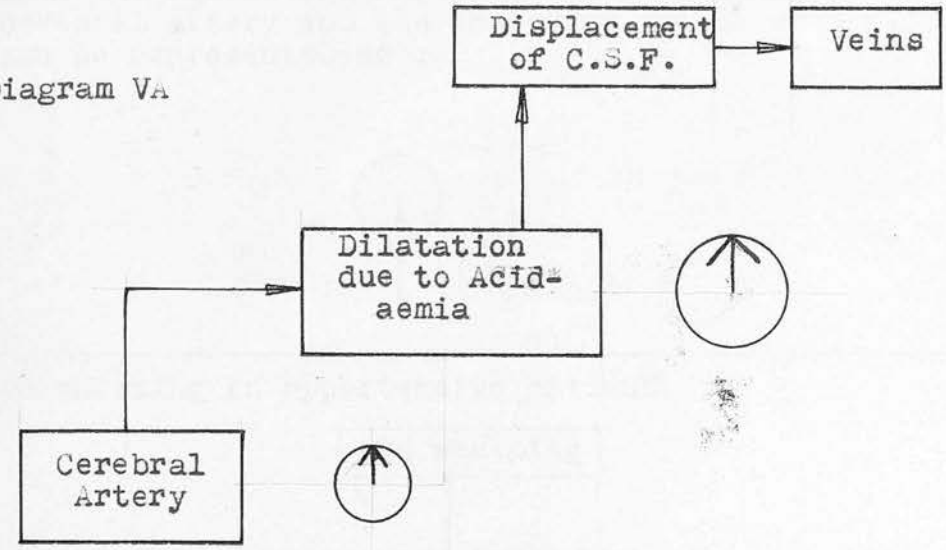


Diagram IV.

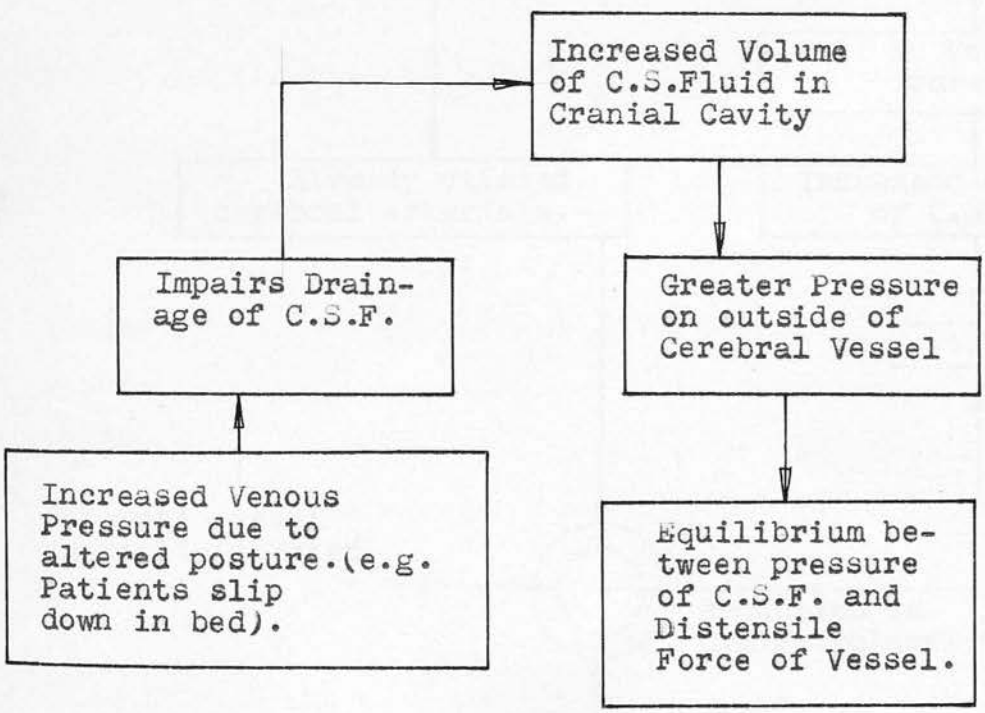

Increased Co_2
Reduced Renal Blood flow
Acidemia
Constant dilatation of Arterioles
Displacement of C.S.F.
Veins

In health during sleep:

Diagram VA

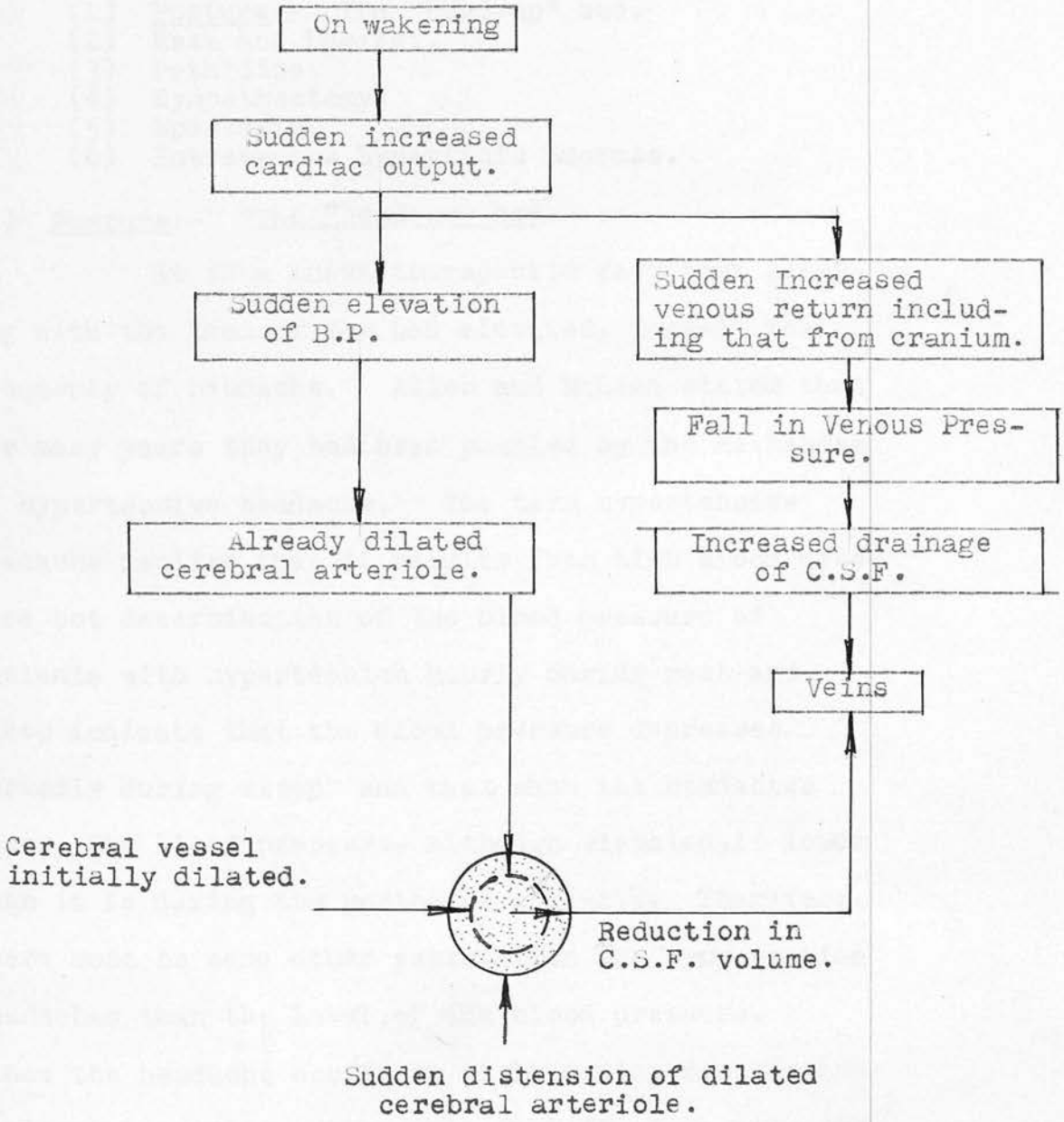


(B) In Hypertension during sleep:



Thus the equilibrium between the dilatation of the cerebral artery and the increased volume of c.c.fluid can be represented so :-

On wakening in hypertensive patient:



(C) Duration of Attack:

Apart from the observation that the attack lasts from half an hour to several hours, no further information can be obtained from this at present.

(D) Methods of relief in these cases :

(1) Posture:- The "head-up" bed.
(2) Rest and Luminal.
(3) Pethidine.
(4) Sympathectomy.
(5) Epistaxis.
(6) Intravenous hypertonic Sucrose.

(1) Posture:- "The "head-up" bed.

It is a known therapeutic fact that sleeping with the head of the bed elevated, lessens the frequency of headache. Allen and McLean stated that for many years they had been puzzled by the mechanism of hypertensive headache. The term hypertensive headache implies that it results from high blood pressure but determination of the blood pressure of patients with hypertension hourly during rest and sleep indicate that the blood pressure decreases markedly during sleep and that when the headaches occur, the blood pressure, although elevated,is lower than it is during the period of activity. Therefore, there must be some other explanation for hypertension headaches than the level of the blood pressure. Since the headache occurs at night during rest in the horizontal position, they felt that it might arise as a result of this horizontal position.

They made an extensive study of this condition and found that the results have been so striking and the effects of positions of the bed so consistent that they are convinced that there is a relationship between the posture which the patient assumed during sleep and the occurrence or absence of headache. Migraine, as well as the hypertension headache, did not occur when the head of the bed was elevated. The "head-up" position is accomplished by elevating the posts of the head of the bed on two 9 inch blocks.

In our series of 5 out of 7 patients with frontal headache (71%), and of 3 out of 5 patients with occipital headache (60%), the frequency of the attacks were lessened by elevation of the head of the bed.

Elevation of the head of the bed will in health:

(1) Reduce venous return from the limbs.
(2) Lessen cardiac output.
(3) Increase venous drainage from the skull.

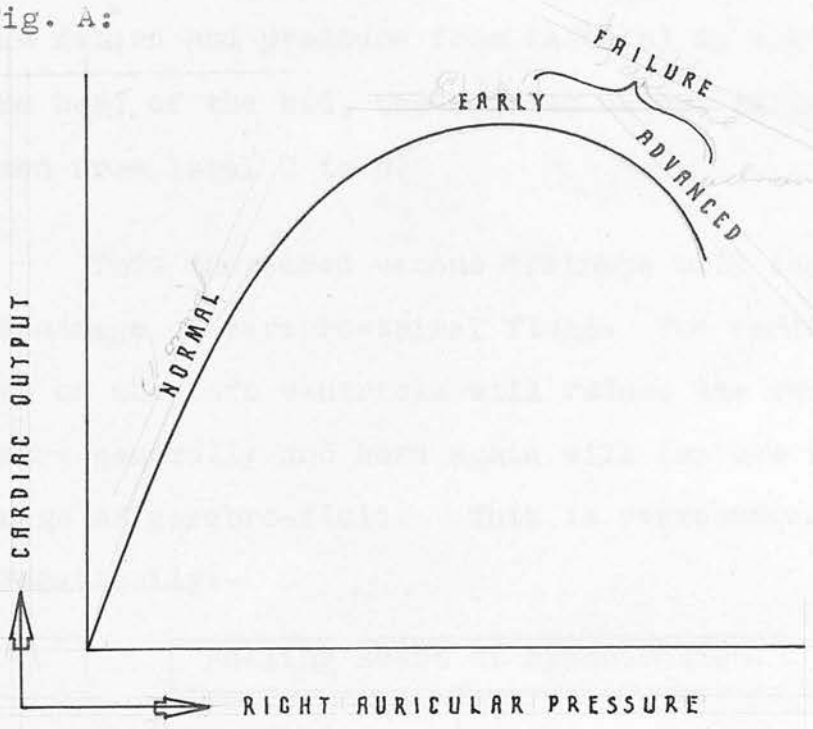
In hypertension with cardiac decompensation, sleeping with the head of the bed raised, will :

(1) Reduce venous return from the limbs.
(2) Reduce right-sided cardiac output and thus lessen the strain on the left ventricle.
(3) Increase venous drainage from the skull.

The effects of elevation of the head of the bed will improve the output of the failing heart although lessen the output of the normal heart for

Starling has shown that :-

Fig. A:



Starling's Curve. Increase of filling pressure is accompanied by an increase in output until the heart is over-loaded; thereafter the output begins to fall with any further increase in pressure (McMichael and Sharpey-Schafer).

FIG II

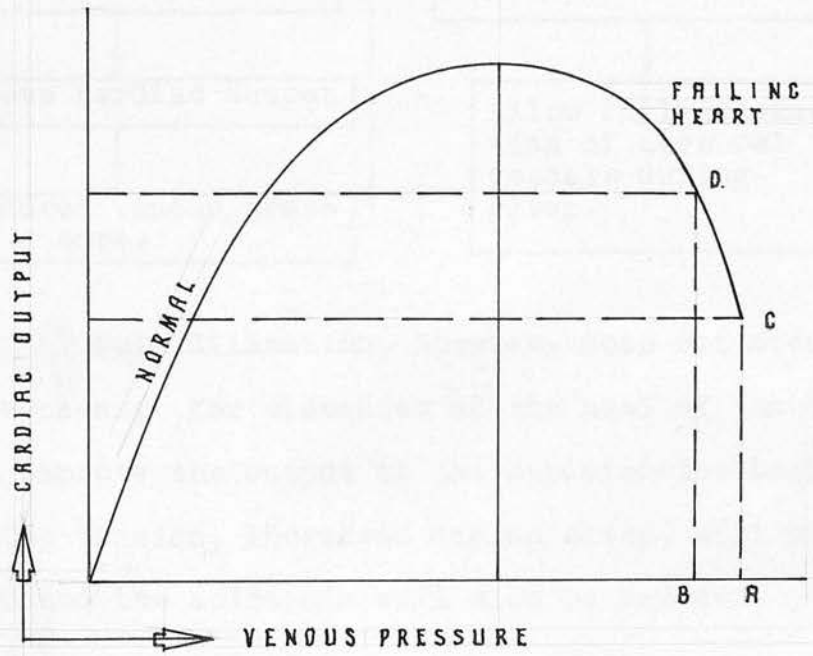
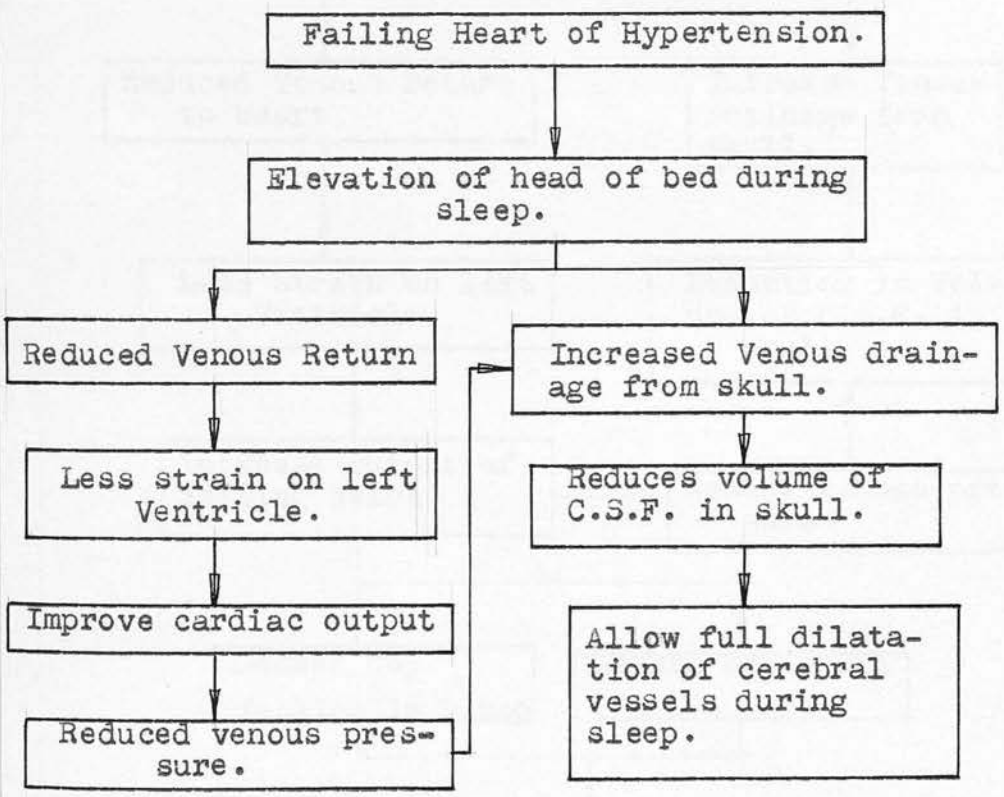


Figure (6) shows that by reduction in venous return and pressure from (A to B) by elevation of the head of the bed, the cardiac output is increased from level C to D.

This increased venous drainage will improve the drainage of cerebro-spinal fluid. The reduced strain on the left ventricle will reduce the venous pressure generally and here again will improve the drainage of cerebro-fluid. This is represented diagramatically:-

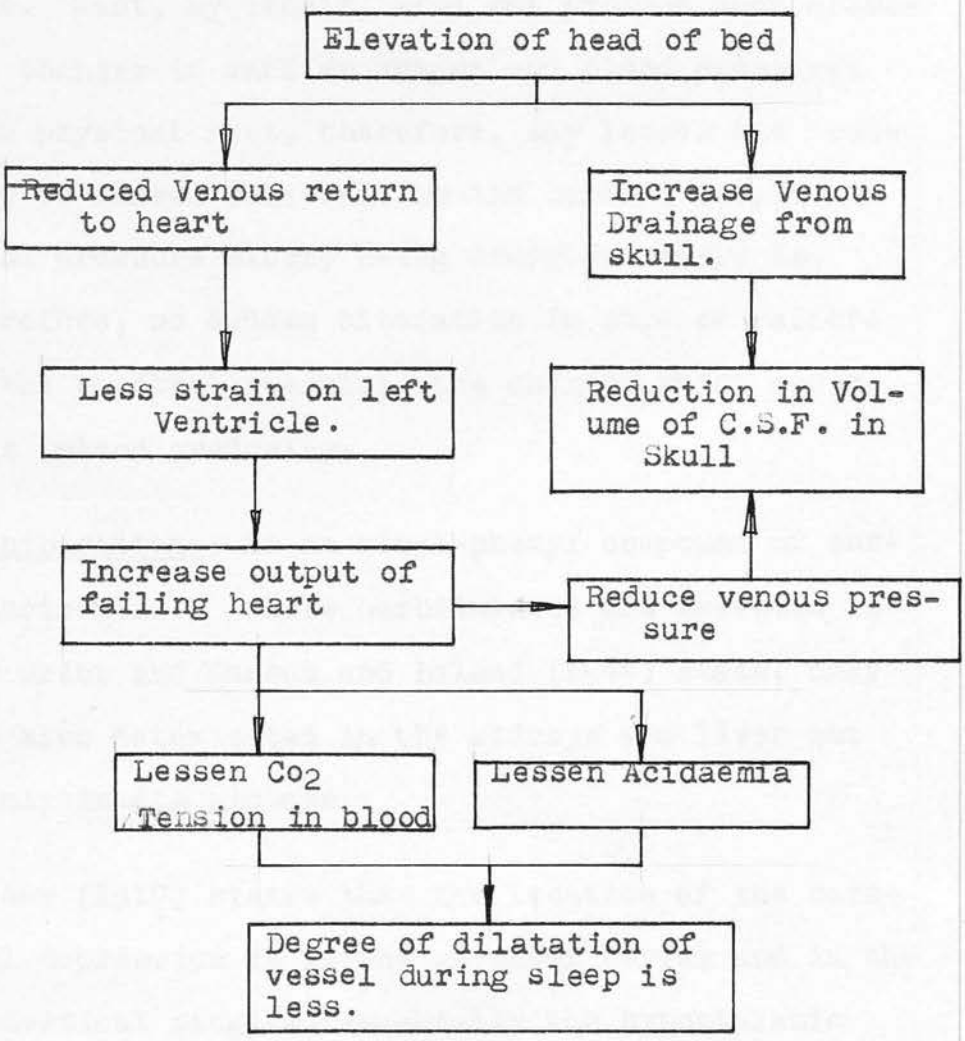


Full dilatation, however, does not occur in these cases; for elevation of the head of the bed will improve the output of the hypertensive heart. The Co_2 tension, increased during sleep, will be reduced and the acidaemia will also be reduced.

The reduction in these two factors will lessen the degree of dilatation of the cerebral vessels during sleep.

By means of elevation of the head of the bed, the degree of dilatation of the cerebral arteries will be less and the drainage of cerebro-spinal fluid will improve. This can be seen diagramatically:-

Effects during sleep:



As we could see from the above diagram in the "head-up" position, the acidaemia and the consequent vascular dilatation is less, the most important basic

factor in the production of the headache is vascular dilatation; so when the patient wakes up, even if there is no apparent change in cardiac output, he does not have headache, because the fundamental factor in its production is missing.

2. Rest and Phenobarbitone:

In patients with frontal headache, relief was obtained in two patients by rest and phenobarbitone. Rest, by itself, will not produce such dramatic changes in cardiac output and blood pressure. Such physical rest, therefore, may lessen the headache or indeed relieve it by the cardiac output and blood pressure slowly being changed. There is, therefore, no sudden alteration in size or calibre of the cerebral vessels; the changes which occur, take place gradually.

Phenobarbitone: is an ethyl-phenyl compound of barbituric acid. These barbiturates are excreted in the urine and Masson and Beland (1944) state, they are also detoxicated in the kidneys and liver but mainly in the kidneys.

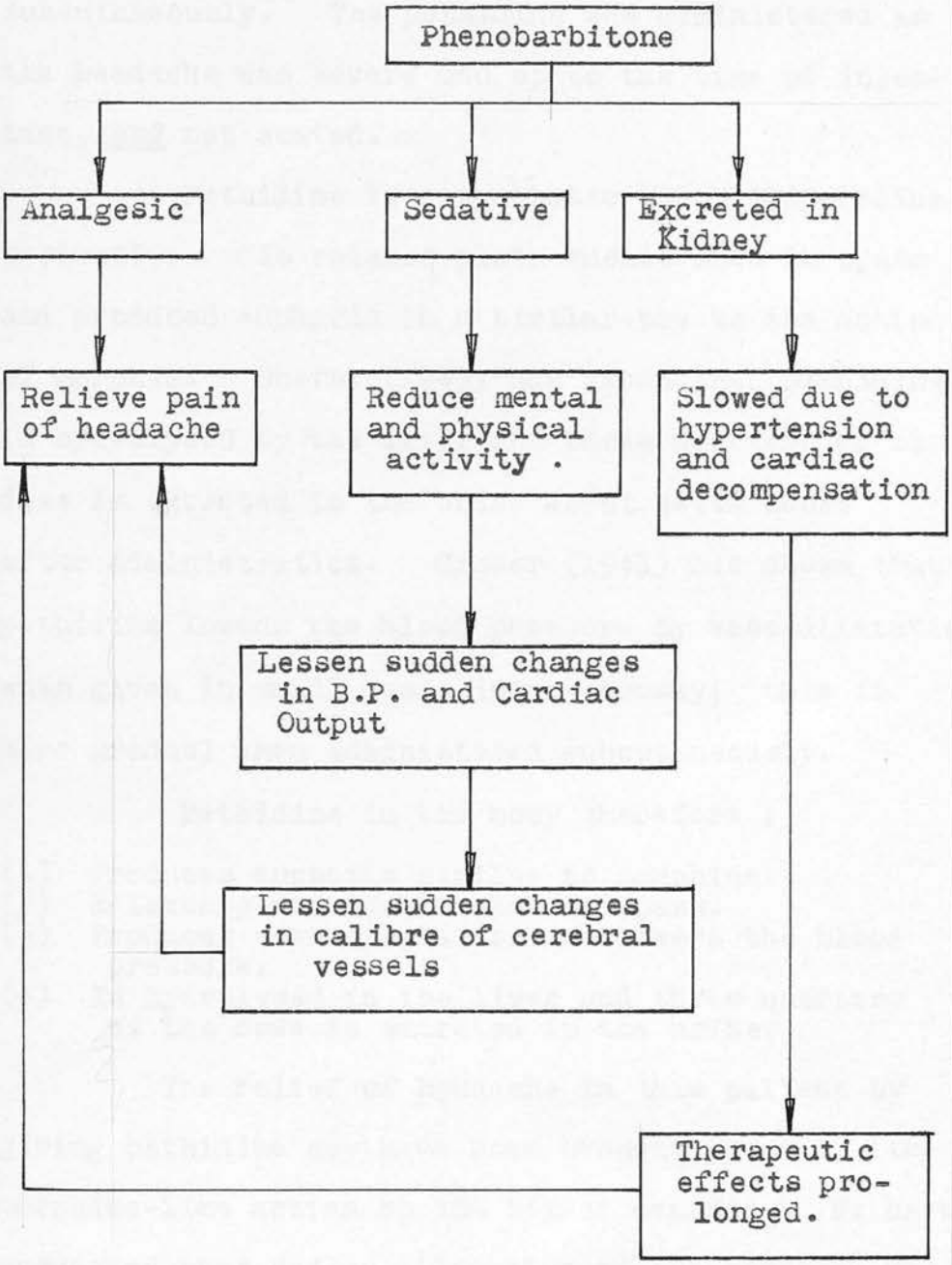
Keeser (1917) states that the location of the cerebral depression is in the cerebral cortex and in the subcortical ganglia, especially the hypothalamic portion of the diencephalon, thus altering the response to pain. Phenobarbitone, therefore, is mainly a sedative but has mild analgesic properties.

When administered to patients with hypertension and especially those with headache then

(1) There is a cortical depression producing sedation.
(2) An action on the nuclei in the diencephalon with resulting mild analgesia.
(3) Prolongation of the above actions due to poor blood flow through the kidney resulting in delayed destruction and excretion if the kidneys are diseased.

The relief, therefore, of the headache following phenobarbitone, is in part, due to the analgesic action, in part due to the sedative effect with gradual changes in cardiac output, just as in physical rest, and the delayed excretion in the urine, augments these actions.

The action of Phenobarbitone can be represented diagramatically :-



(3) Pethidine:

One patient with frontal headache obtained relief by the administration of 100 mgms of pethidine subcutaneously. The pethidine was administered as the headache was severe and up to the time of injection, had not abated.

Pethidine is a synthetic phenylipiperidine derivative. It relaxes plain muscle when in spasm and produced euphoria in a similar way to the action of morphia. Oberst (1943) has shown that pethidine is hydrolysed by the liver and three quarters of its dose is excreted in the urine about seven hours after administration. Gruber (1941) has shown that pethidine lowers the blood pressure by vaso-dilatation when given in small doses intravenously; this is more gradual when administered subcutaneously.

Pethidine in the body therefore :

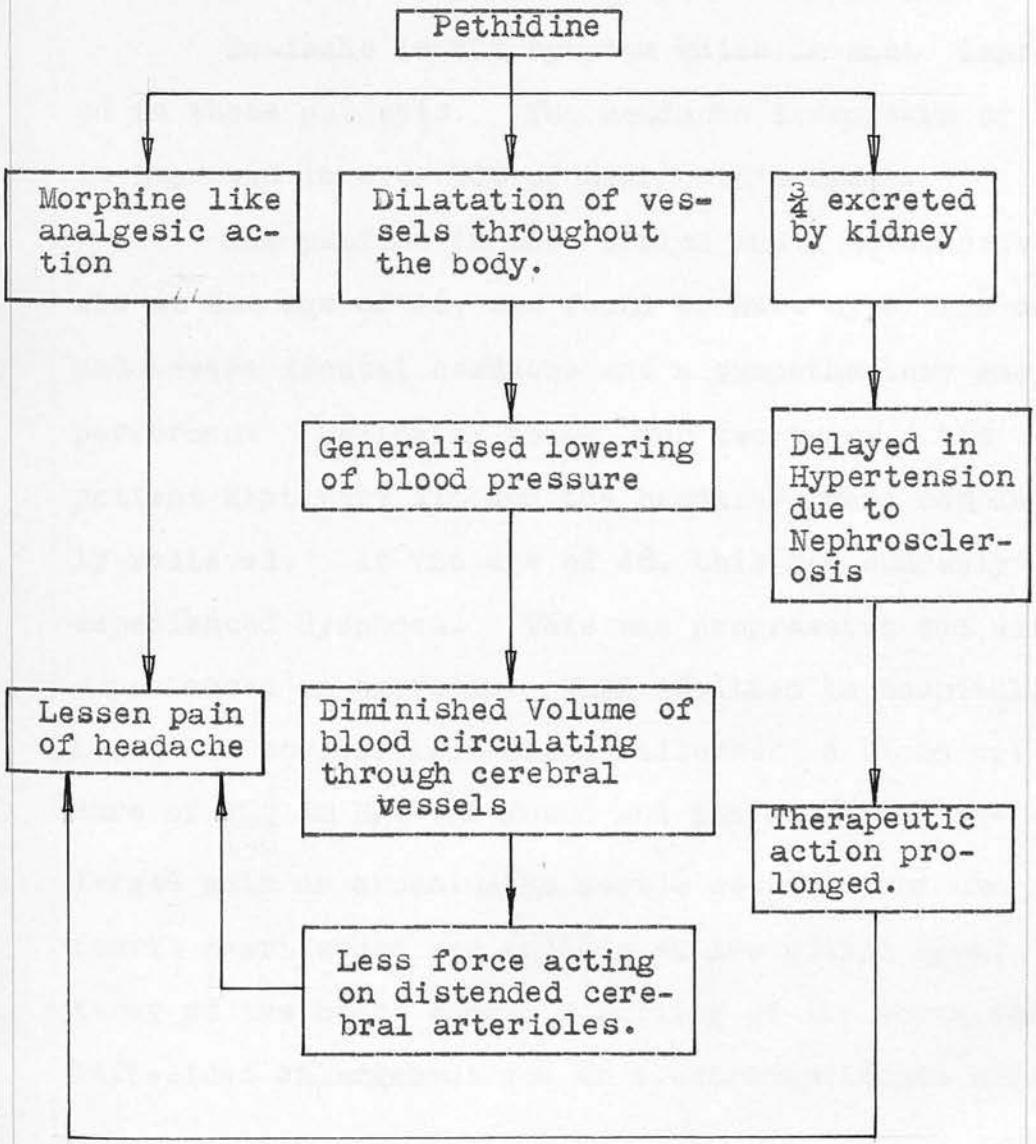
(1) Produces euphoria similar to morphine.
(2) Relaxes plain muscle when in spasm.
(3) Produces vaso-dilatation and lowers the blood pressure.
(4) Is hydrolysed in the liver and three quarters of the dose is excreted in the urine.

The relief of headache in this patient by giving pethidine may have been brought about by its morphine-like action on the higher centres. We have suggested that sudden dilatation of the cerebral vessels produces headache. Pethidine by its action on plain muscle will lower the blood pressure and it produces vaso-dilatation throughout the body. The lowering of the blood pressure by this generalized

vaso-dilatation will lessen the degree of distension which these vessels are subjected to after the patient wakens and the blood pressure is suddenly raised.

Furthermore, the delayed excretion by the kidney will augment the above actions provided the kidney function is impaired.

The actions of Pethidine represented diagramatically:



(4) Sympathectomy:

Learmonth (1950) has stated that the indications for sympathectomy in hypertension are limited. It is as wrong to deny to sympathectomy, a place in the symptomatic treatment of this condition as it is to claim for sympathectomy a uniformly favourable influence upon its symptoms and progress. The risks of the operation should be minimal and for this the scope of the operation should be restricted.

Learmonth maintains that for relief of symptoms a subdiaphragmatic operation may be sufficient.

Headache is the symptom which is most improved in these patients. The headache disappears or is improved in over 90% of Learmonth's cases.

The patient in this series was a motor-driver, who at the age of 46, was found to have hypertension and severe frontal headache and a sympathectomy was performed. Following this, for two years, the patient kept very fit and the headaches were completely relieved. At the age of 48, this man suddenly experienced dyspnoea. This was progressive and was experienced on exertion. When admitted to hospital, he was in obvious left-sided failure. A blood pressure of $\frac{208}{130}$ mm Hg. was found and the heart was enlarged with an accentuated aortic second sound and a fourth heart sound was audible at the mitral area. X-ray of the heart showed unfolding of the aorta and left-sided enlargement and an electrocardiogram showed

marked left axis deviation. His urine findings were normal and the blood urea nitrogen was 18 mgm%. The fundi showed grade II. changes.

The patient responded well to rest, aminophylline and phenobarbitone and his blood pressure rose to $\frac{240}{130}$ mm Hg. with recovery from the heart failure. A sodium amytal test was performed and revealed that the blood pressure was not labile. Urea range showed evidence of early renal impairment. At no time in hospital did the patient experience any headache.

The explanation of the failure of return of headache, in spite of the blood pressure rising, is difficult. The Smithwick operation results in generalised splanchnic dilatation.

Theories for Operation: (Learmonth, 1947).

(1) The hypothesis that by interrupting the vaso-constrictor nerves to the splanchnic area, vaso-dilatation would be produced in a large area which would result in a fall of blood pressure.

(2) The hypothesis that by interrupting the nerves to the adrenal glands, the output of adrenal be reduced.

(3) The hypothesis that by interrupting the nerves to the renal vessels, renal ischaemia will be diminished as a consequence of vaso-dilatation.

The effect of sympathectomy, therefore is to increase splanchnic dilatation. This acts as a reservoir for the circulating blood and thus reduces the venous pressure and therefore there is no

engorgement of the neck veins nor impaired venous or cerebro-spinal fluid drainage from the skull. The output of the heart tends to be reduced and therefore there are not such sudden changes in cardiac output and blood pressure levels nor in cerebro-spinal fluid volume and pressure on rising, with resulting sudden over-distension of the cerebral arterioles, and the headache, therefore, should not occur or at least be improved. (See diagram 2).page 127

Why the headache does not return, when the blood pressure rises again, and there is heart failure as in this case, is not known. But presumably, this can be likened to sleeping in the "head-up" position. There is in fact, due to splanchnic dilatation, reduced venous return with improvement of the cardiac output and the effects on the cerebral vessels are just as described previously on wakening from the "head-up" position.

Furthermore, the splanchnic dilatation improves the circulation to the kidney. Thus the acidaemia is less and there is less dilatation of the cerebral vessels.

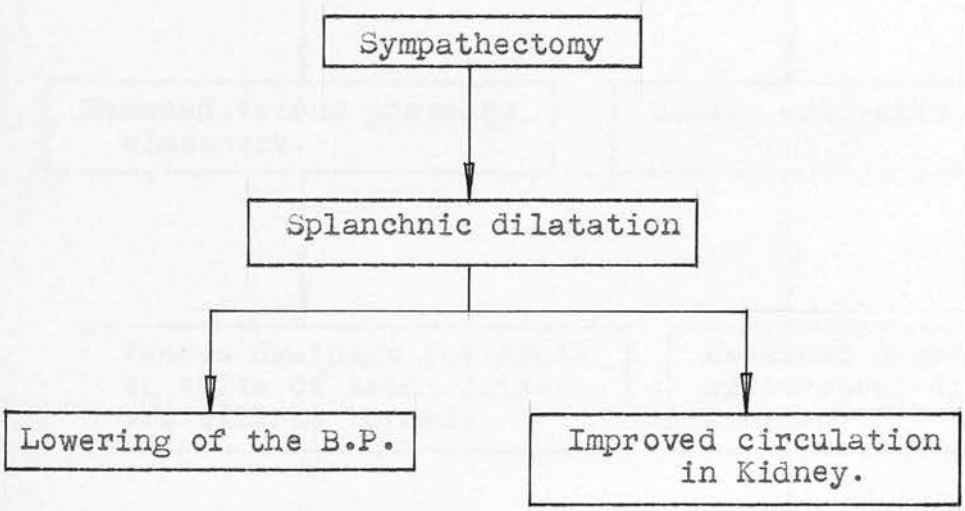
The heart failure present in this man did not cause a return of the headache, for it can be assumed, that the increased venous pressure resulting from the failure was mainly splanchnic, due to the sympathectomy, and there would be very little congestion of the cerebral vessels.

The above is the only explanation that can

be suggested, for the failure of the headache to return in spite of the return of blood pressure to its high level has puzzled many.

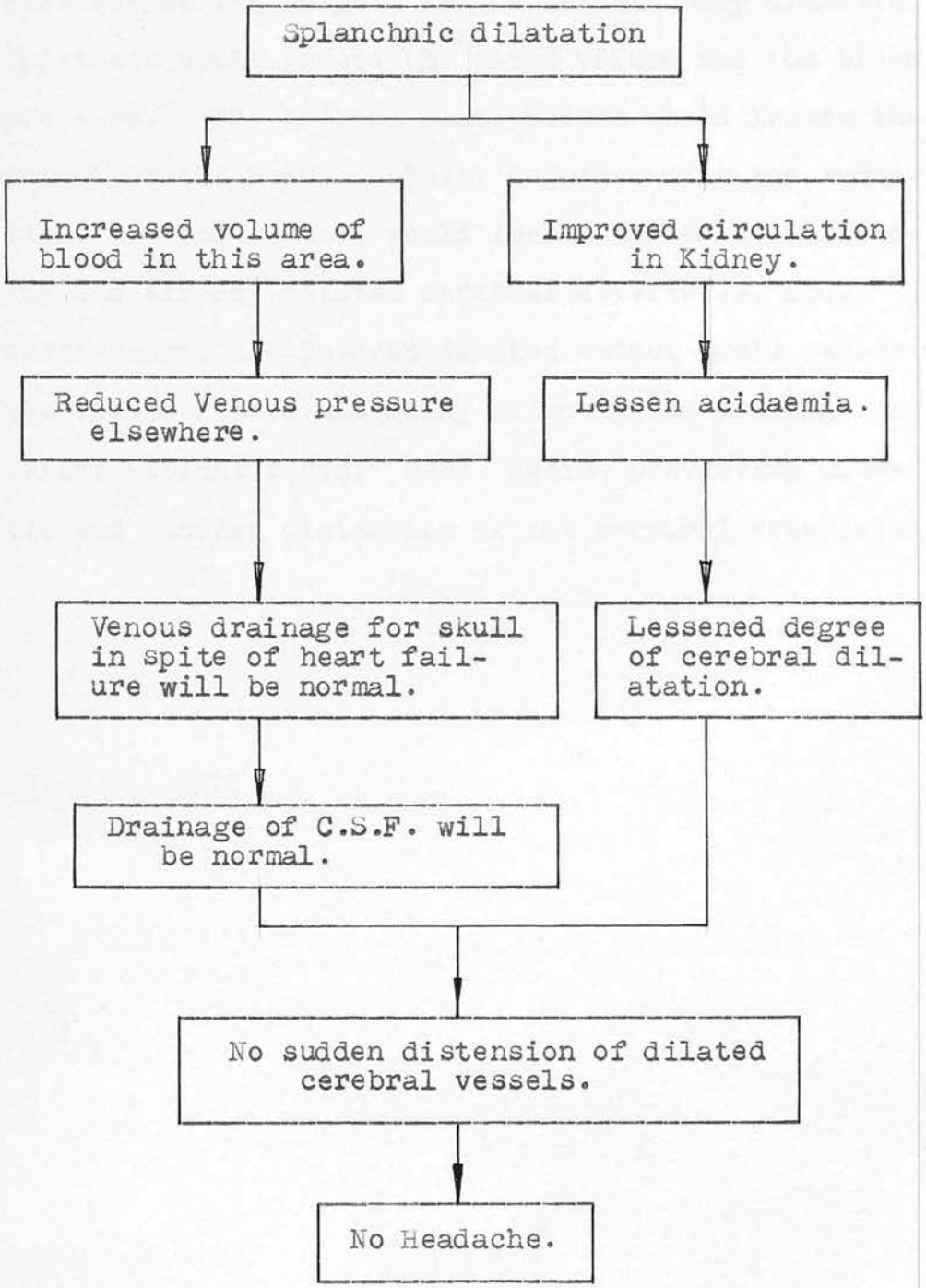
The effects of Sympathectomy represented diagramatically :

Diagram I.



Suggested Method of relief of Headache :

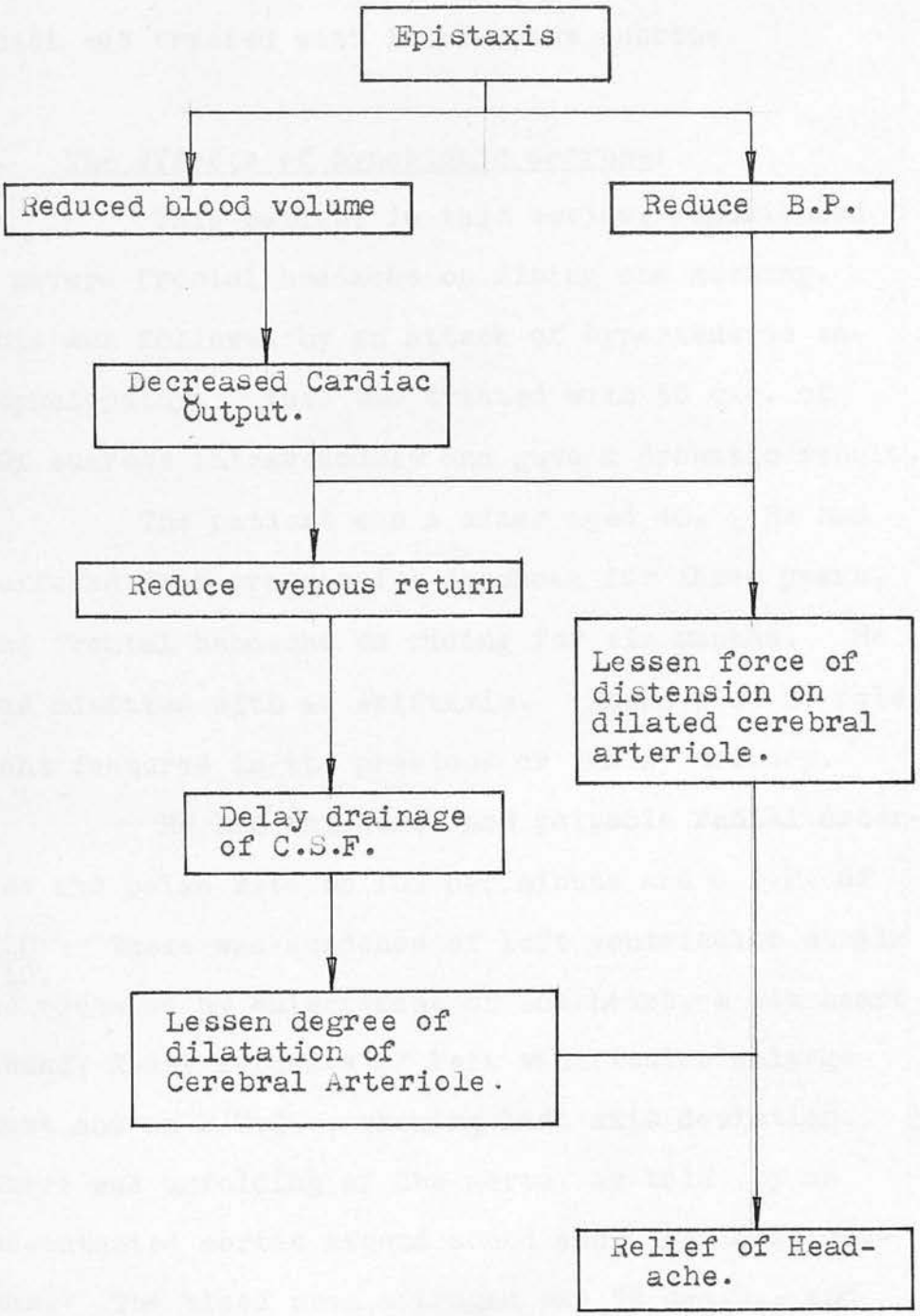
Diagram 2.



5. Epistaxis :

One patient obtained relief from the headache, when his nose began to bleed. At this time, the headache was at its height, and relief was very dramatic. Epistaxis would reduce the blood volume and the blood pressure. The reduced blood volume would lessen the output of the heart. This, together with the reduction in blood volume, would lessen the force distending the already dilated cerebral arterioles, and, furthermore, the lowered cardiac output would reduce the venous return and delay slightly the drainage of cerebro-spinal fluid; here again, preventing drastic and sudden distension of the cerebral arterioles.

The effects of Epistaxis represented diagramatically:



One of the seven patients, suffering from frontal headache, who was in hospital, receiving treatment developed an attack of hypertensive encephalopathy, which was treated with intravenous sucrose.

6. The effects of hypertonic sucrose:

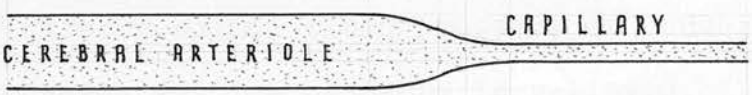
This patient in this series, experienced a severe frontal headache on rising one morning. This was followed by an attack of hypertensive encephalopathy. This was treated with 50 c.c. of 50% sucrose intravenously and gave a dramatic result.

The patient was a miner aged 40. He had suffered from progressive dyspnoea for three years, and frontal headache on rising for six months. He was admitted with an epistaxis. There were no relevant features in the previous or family history.

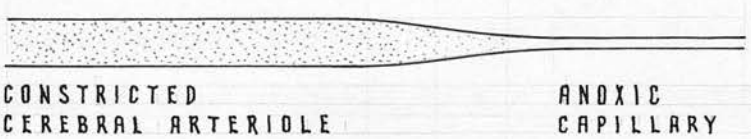
He had thickened and palpable radial arteries and pulse rate of 100 per minute and a B.P. of $\frac{220}{110}$. There was evidence of left ventricular strain as revealed by enlargement of the heart, a 4th heart sound, X-Ray evidence of left ventricular enlargement and an E.C.G.., showing left axis deviation. There was unfolding of the aorta, as told by an accentuated aortic second sound and a systolic murmur. The blood urea nitrogen was 58 mgm.%., and the urinary findings were a specific gravity of 1018, albumin and hyaline casts and a urea range of sp. gr. 1018 and 2 grammes per cent - 1003 and 1 gramme per cent. The fundi were stage III.

Although there was no papilloedema, the age, blood pressure level, urinary findings and presence of hypertensive encephalopathy would place the patient in the malignant hypertensive group or would suggest that he would develop malignant hypertension.

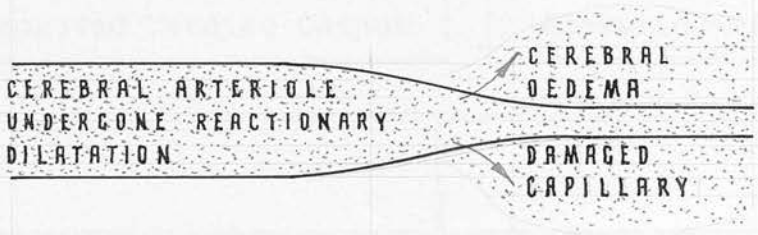
The mechanism of fits in hypertension has been explained by an unstable state of the cerebral arterioles prior to the state of arteriolonecrosis. They respond to changes in blood pressure by sudden constriction followed by sudden distension and exudation of fluid into the brain substance from the distal capillaries; thus resulting in cerebral oedema. This can be seen diagramatically:



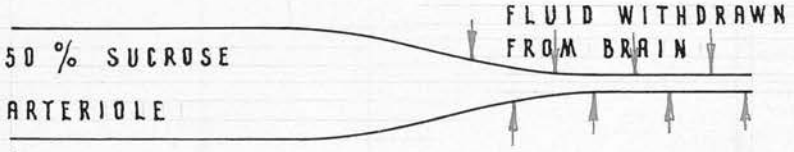
Sudden elevation of blood pressure acting on unstable cerebral arterioles produces constriction with resulting anoxaemia of distal capillary wall.



Sudden release of spasm and dilatation of cerebral arteriole resulting in exudation of fluid through damaged capillary, giving cerebral oedema and fits.

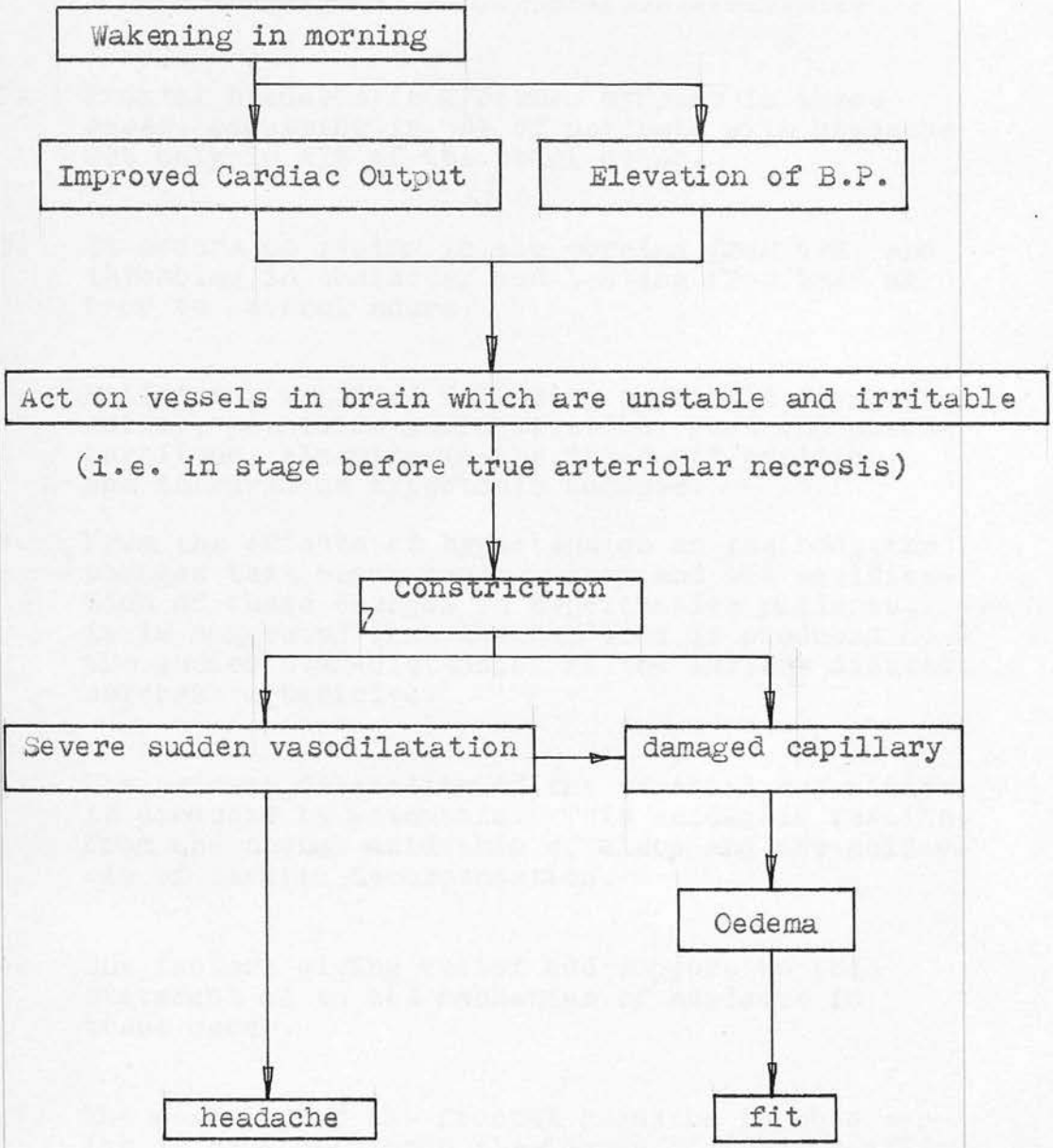


The giving of hypertonic sucrose raises the osmotic pressure of the blood and thus excess cerebro-spinal fluid producing the oedema is withdrawn back into the capillaries and circulation; and thus the cerebro-spinal fluid pressure is reduced, and the fits cease, and the headache is relieved. Diagramatically :



The patients with hypertensive encephalopathy invariably experience an excruciating headache which is immediately followed by the fit. The time relationship of this headache to the sequence of events is as follows, when the cerebral arterioles undergoes its reactionary extreme vaso-dilatation there is headache following the period of constriction, and the oedema results in the fit.

Diagramatically:



Preliminary Conclusions based on Patient with Frontal Headache in Essential Hypertension.

1. Frontal headache is a common symptom in these cases, occurring in 58% of patients with headache but only in 47% of the total group.

2. It occurs on rising in the morning from bed, and throbbing in character and lasting from half an hour to several hours.

3. Relief was obtained following epistaxis, sympathectomy, pethidine administration, rest and phenobarbitone, sleeping in the "head-up" position, and intravenous hypertonic sucrose.

4. From the effects of hypertension on the body, the changes that occur during sleep and the modification of these changes in hypertensive patients, it is suggested that the headache is produced by the sudden over-distension of the already dilated cerebral arterioles.

5. The primary dilatation of the cerebral arterioles is produced by acidaemia. This acidaemia results from the normal acidaemia of sleep and the acidaemia of cardiac decompensation.

6. The factors giving relief add support to this statement as to the mechanism of headache in these cases.

(7) The severity of the frontal headache in this series is dependent upon the degree of cardiac efficiency, height of the blood pressure (systolic and diastolic) and fundal changes.

B. Dealing with the Occipital Headache:

When the occipital headache is considered, then similar factors play a part.

It is known that there is a relationship between the blood urea nitrogen and the fundal changes, and the level of the blood pressure and the blood urea nitrogen. Furthermore, the severity of the headache is related to the level of blood urea nitrogen, fundal changes and systolic pressure.

As before, headache was experienced on rising from bed in the morning, and the relief was obtained by:-

(1) Rest and phenobarbitone in two cases.
(2) Epistaxis and Venesection in one case.
(3) Aspirin in " "
(4) No relief in " "
(5) The "head-up" position in three cases.

These factors will now be considered in the light of the changes present in the body.

(1) Sleep: (Kleitman). The same mechanism occurs here as it does in patients with frontal headache.

(I.) There is a fall in blood pressure.
(II) A decrease in heart rate.
(III) Respiratory rate is not changed.
(IV) Co_2 tension in alveolar air and arterial blood is
(V) raised due to depression of respiratory centre.
(VI) B.M.R. is lowered.
(VII) Intestinal tone is unaffected.
(VIII) There is a smaller volume of urine secreted.
(IX) and thus there is a mild acidaemia
(X) Radiation of skeletal muscle.

(2) Hypertension: Here again, the same factors are present as in the cases with the frontal headache

and are modified by sleep:

(I) Reduced cardiac output.
(II) Increased Co_2 tension in the blood.
(III) Acidaemia.

Because the renal impairment seems to play an important rôle in the severity of the headache, it will be considered in more detail; for the obvious difference between the patients with frontal and occipital headache in this series, is, that the cases with occipital headache have a greater elevation of the blood/nitrogen and show an evidence of renal impairment. /urea

The effects of renal impairment in the body are many. Fishberg (1939)(a) states that when the accomplishment of the kidneys falls behind the needs of the organism, potential urinary constituents accumulate in the blood. Among the potential urinary constituents are always nitrogenous end-products of protein katabolism, with resultant rise in the non-protein nitrogen of the blood.

In recent years, the development, particularly by American investigators, of accurate analytical methods requiring only small quantities of blood, has led to the accumulation of much data concerning the numerous and diverse changes in the chemical composition of the blood produced by renal insufficiency. These changes are brought about in four general ways, the relative importance of which varies in different circumstances.

1. Retention in the blood of those potential urinary constituents for which the extra-renal avenues of elimination are inadequate even when functioning vicariously. These belong to three groups.

   (a) Because the extra-renal paths of excretion for the nitrogenous end-products of protein Katabolism are quantitatively inadequate, and break down of protein goes on to some extent whatever the diet, renal retention is always marked by increase in non-protein nitrogen of the blood. Likewise, the phosphate and sulphate resulting from the oxidation of the amino-acids and other substances containing phosphorus and sulphur are retained.

   (b) When chloride and other substances excreted predominantly by the kidney, are ingested in amounts exceeding the extra-renal loss (including that by vomiting), they accumulate in the organism.

   (c) Finally, indican, phenols, and the products of intestinal putrefaction which are normally absorbed from the gut and excreted in the urine may also be retained.

2. The retained urinary constituents may cause secondary changes in the chemical composition of the blood. Thus phosphate retention tends to lower the calcium content of the blood and retention of fixed acid depresses the blood bicarbonate.

3. Tubular insufficiency results in failure adequately to reabsorb water and various electrolytes, notably sodium and chloride, with consequent tendency to dehydration and demineralization of the blood and tissues.

4. Failure of the synthetic function of the kidney results in loss of fixed base, which is excreted in conjunction with acid ions in place of the ammonia that the kidney is unable to form.

Urea accumulates in the blood and is an indication of the efficiency of the kidney and the circulation through the kidney. Urea, however, has been shown to be non-toxic. It is only important being an indication of renal efficiency.

Acidosis is present in these cases. It is due to:

(1) Retention of inorganic ions.
(2) Diminished ammonia formation by Kidney.
(3) Diminished calcium ion content of the blood.
(4) Retention of organic acids in the blood.

In kidney insufficiency acidosis is the most marked change which is injurious to the body. The body changes in renal failure are due to a complex autointoxication, being the summation of many factors, viz. retention of nitrogenous substances, changes in the osmotic pressure, dehydration, and salt depletion and acidosis. Alteration in the osmotic pressure, dehydration and salt depletion and retention of nitrogenous substances do not affect the blood vessels. Acidosis, however, produces a marked vaso-dilatation. The alteration in osmotic and salt depletion, however, produce changes in the blood volume and the retention of nitrogenous substances produce depression of cellular activity.

In renal failure, the following effects in the body are:-

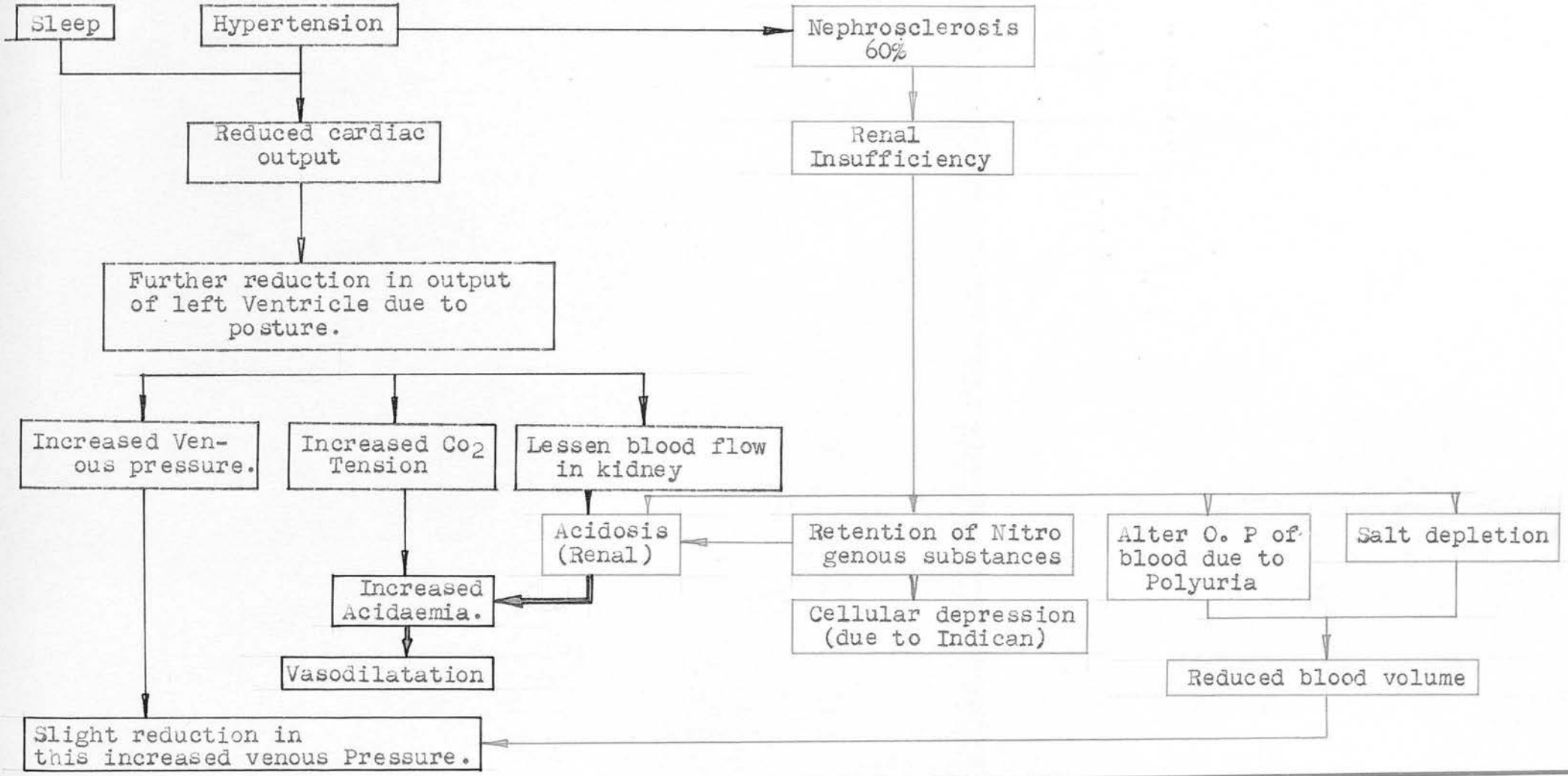
(A) Acidosis producing vaso-dilatation.
(B) Alteration in osmotic pressure.
(C) Salt depletion. Both (B) and (C) reduce the blood volume.
(D) Retention of nitrogenous substances depressing cellular activity.

Renal Impairment and changes in the body during sleep:

When the above factors are considered in the presence of the bodily changes in hypertension during sleep, we find that the acidosis of the renal insufficiency will further increase the degree of vaso-dilatation. The reduction in Blood Volume, due to the

salt depletion, will tend to lessen the increase in venous pressure resulting from the effect of sleep in the hypertensive patient. Indican retention depresses all body cells. This can be represented diagramatically:-

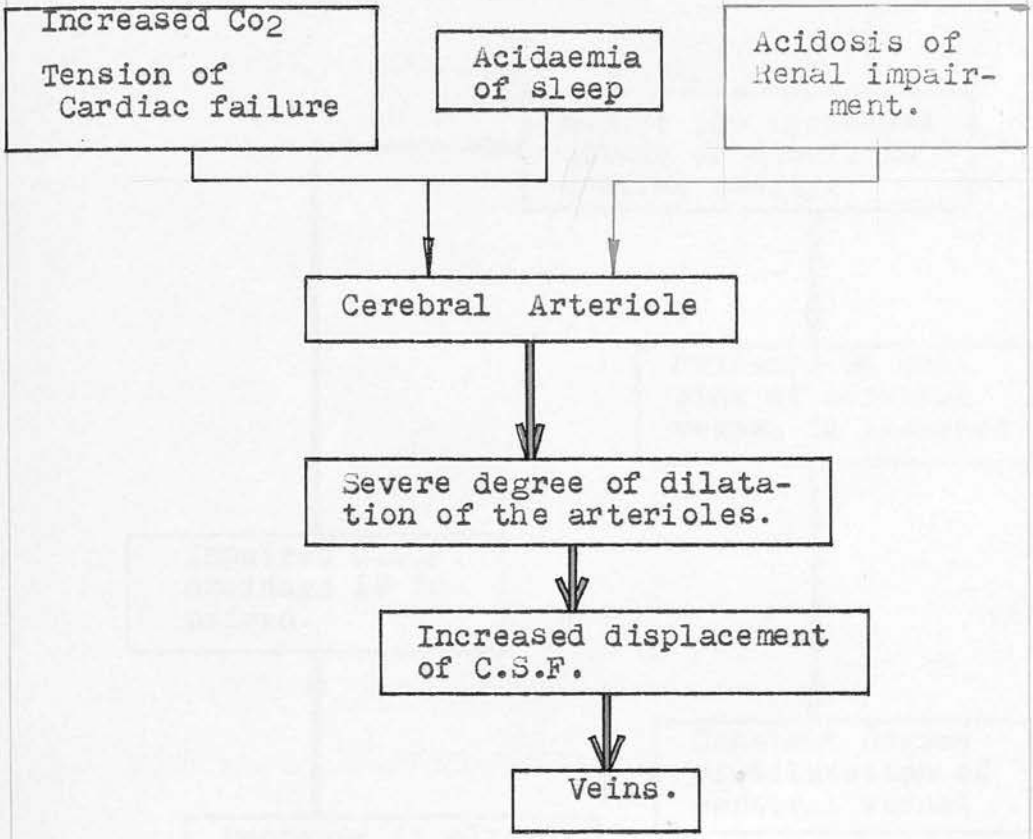
Effects of sleep in hypertensive patient with renal impairment in body generally.



The increased acidosis producing vasodilatation,will automatically, increase the degree of dilatation of the cerebral vessels. The reduction in the blood volume tending to lessen the venous pressure will, therefore, increase the drainage of the cerebro-spinal fluid, displaced by the greatly dilated vessel. We thus have a state of almost complete dilatation of the cerebral vessels. The drainage of cerebro-spinal fluid, although still not normal due to the heart failure, is increased, compared to the state of pure cardiac failure with no renal involvement.

On wakening, therefore, the same mechanism holds as in the patients with frontal headache. The cardiac output increases, the cerebro-spinal fluid drainage is further increased and the resulting elevation of the blood pressure will produce further distension of the already dilated cerebral arterioles. Here again the changes may be represented diagramatically :-

Effects of Sleep in hypertensive patients with Renal impairment on cerebral circulation:



Effects of Sleep during Health:

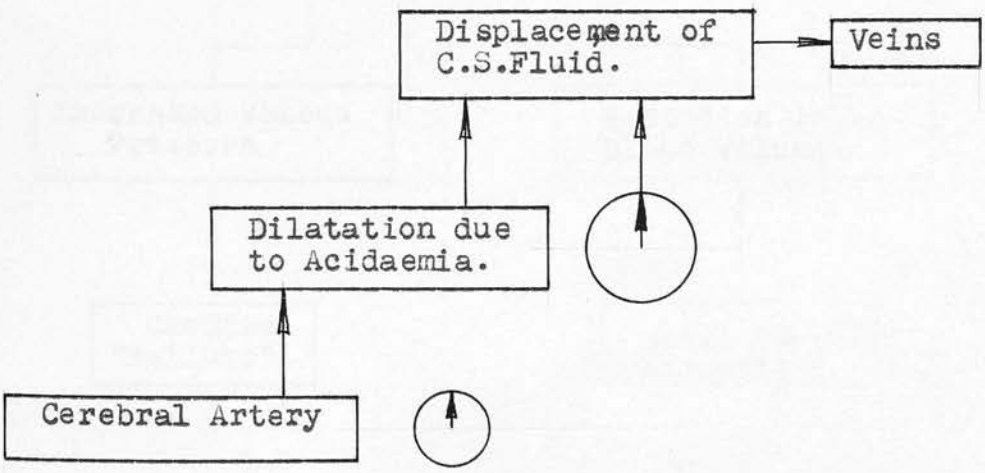
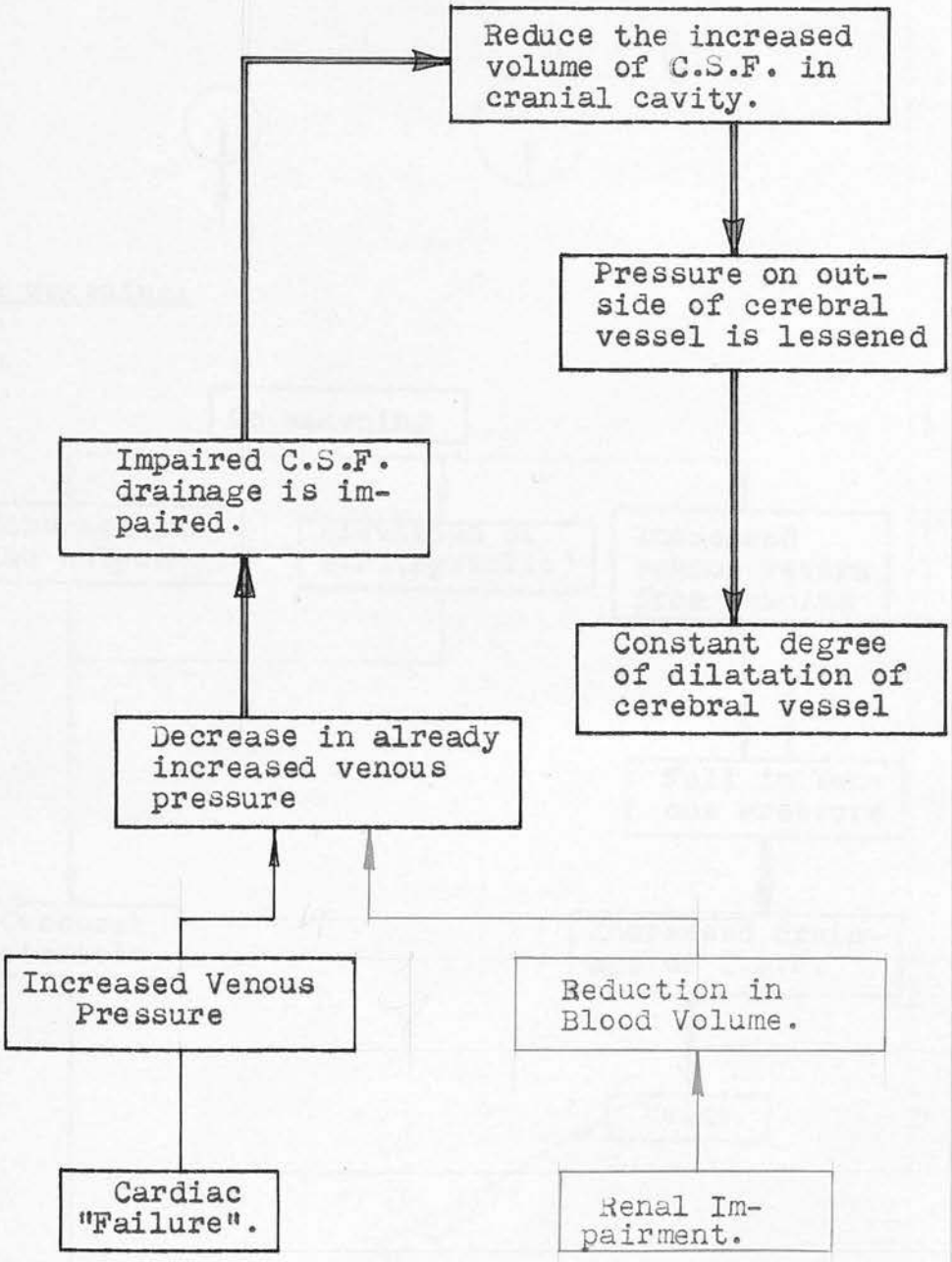
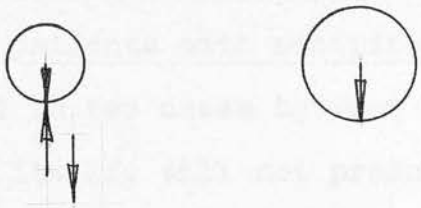


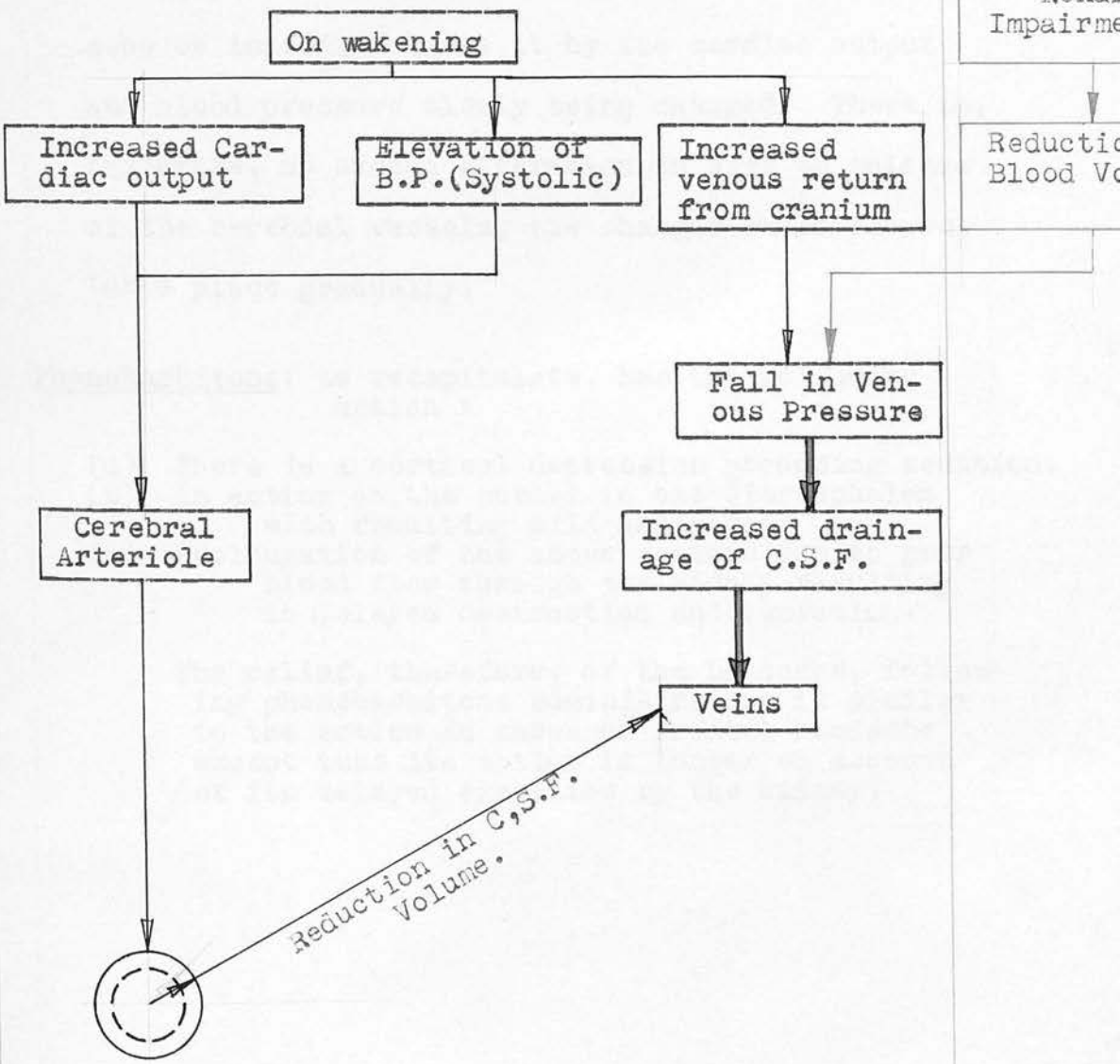
Diagram (8) can be further elaborated.



The equilibrium between the cerebro-spinal fluid and the distensile force in the artery is disturbed - the distensile force being greater than the cerebro-spinal fluid pressure. Thus:-



On wakening:



B. Methods of Relief:

1. Rest and phenobarbitone.
2. Epistaxis and Venesection.
3. Aspirin.
4. "Head-up" position.

1. Rest and Phenobarbitone:

In patients with occipital headache, relief was obtained in two cases by rest and phenobarbitone.

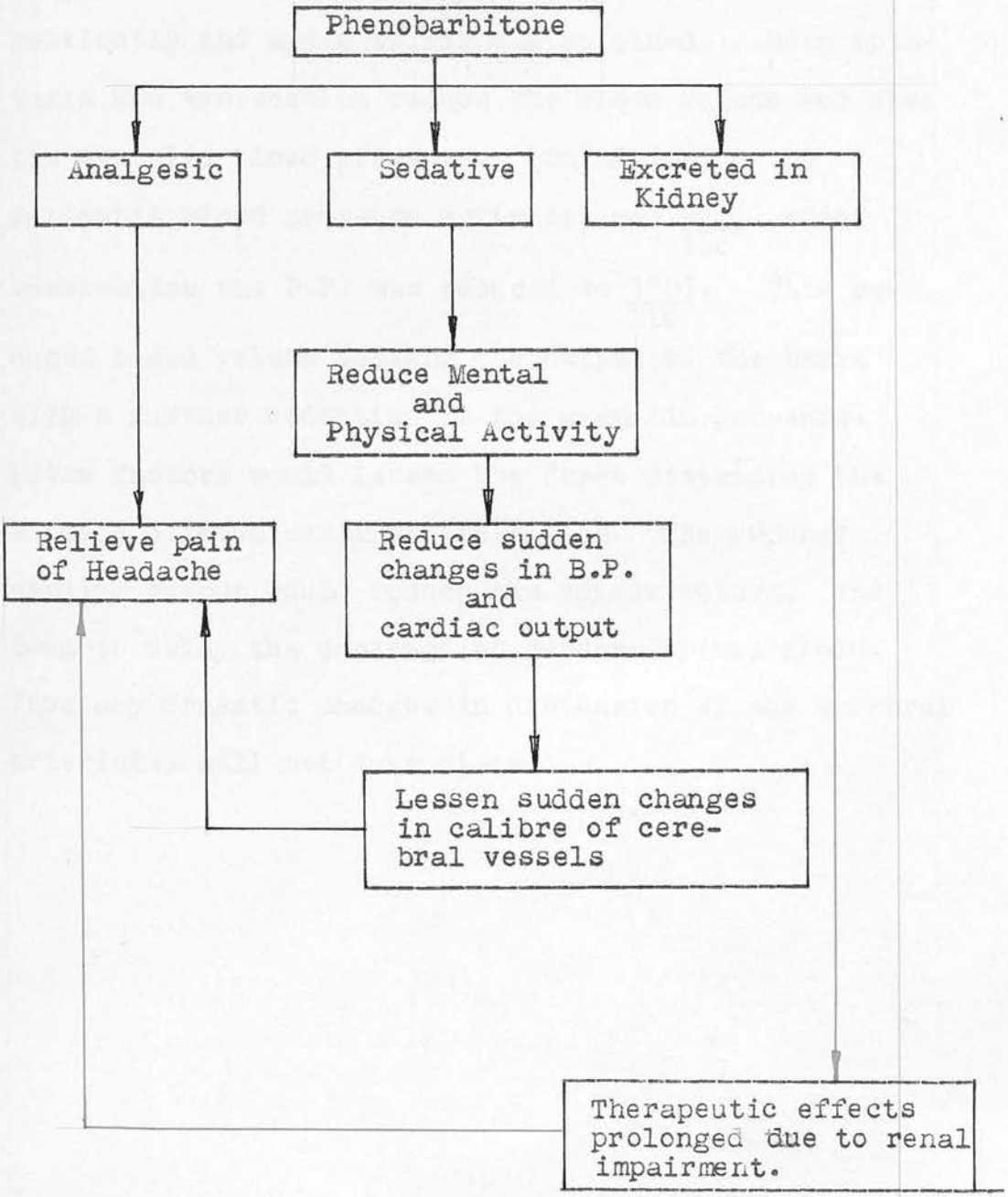
Rest, by itself, will not produce such dramatic changes in cardiac output and blood pressure. Such physical rest, therefore, may lessen the headache or indeed, relieve it by the cardiac output and blood pressure slowly being changed. There is, therefore, no sudden alteration in size or calibre of the cerebral vessels, the changes which occurs, takes place gradually.

Phenobarbitone: to recapitulate, has the following action :

(a) There is a cortical depression producing sedation.
(b) An action on the nuclei in the diencephalon with resulting mild analgesia.
(c) Prolongation of the above actions due to poor blood flow through the kidney resulting in delayed destruction and excretion.

The relief, therefore, of the headache, following phenobarbitone administration is similar to the action in cases of frontal headache except that its action is longer on account of its delayed excretion by the kidney.

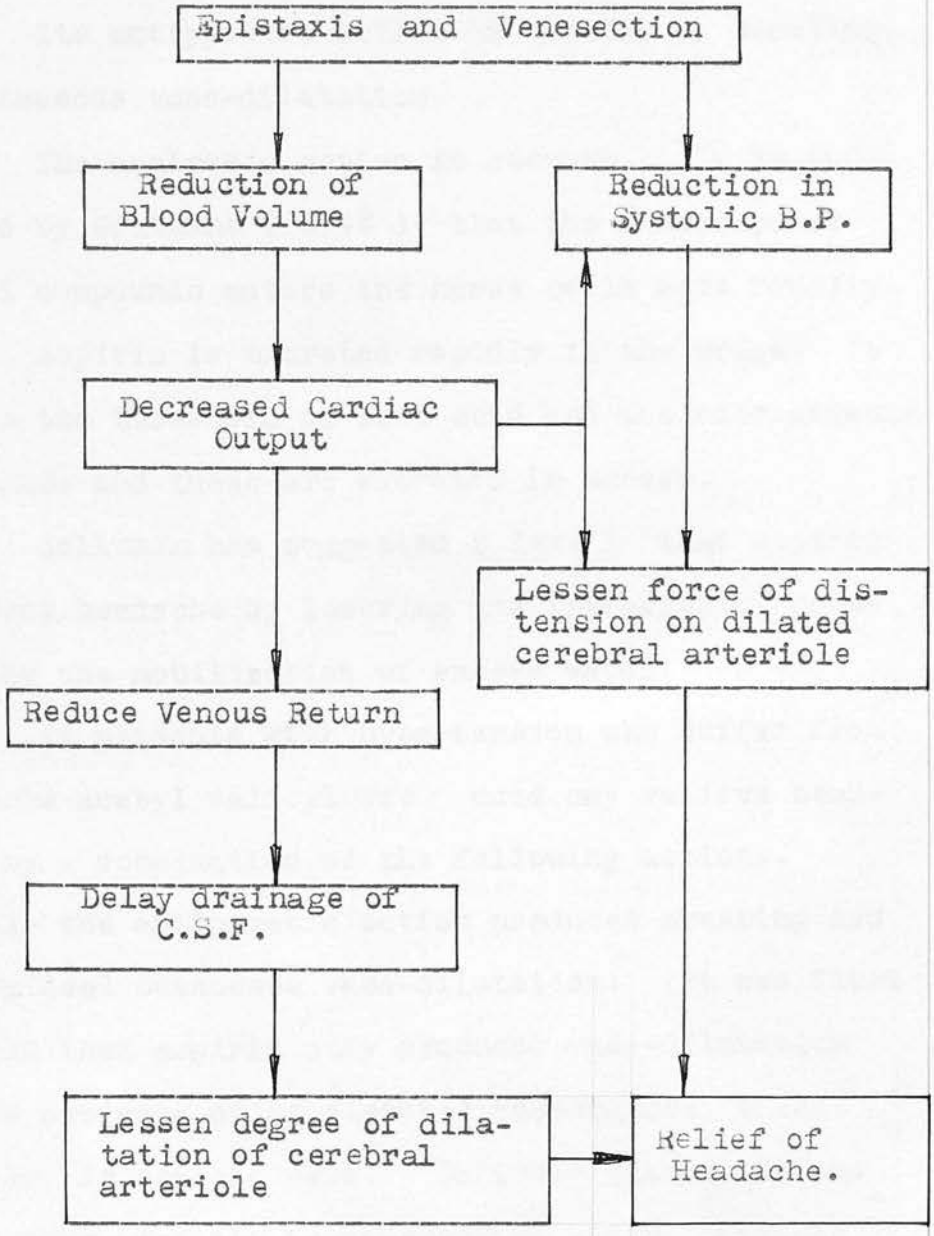
The action of phenobarbitone represented diagramatically :



2. Epistaxis and Venesection:

One patient obtained relief from the headache when his nose began to bleed (he has had this for the past eight years). Venesection was performed therapeutically and again relief was obtained. Both epistaxis and venesection reduce the blood volume and also the systolic blood pressure. (On admission the patient's blood pressure estimated was $\frac{220}{120}$; after venesection the B.P. was reduced to $\frac{170}{110}$). This reduced blood volume lessens the output of the heart with a further reduction in the systolic pressure. These factors would lessen the force distending the already dilated cerebral arterioles. The reduced cardiac output would reduce the venous return, and tend to delay the drainage of cerebro-spinal fluid. Thus any dramatic changes in distension of the cerebral arterioles will not take place.

The effects of Epistaxis and Venesection represented diagramatically :-



4. Aspirin:

This gave relief in one case. Aspirin was introduced by Dreser in 1899. It is used extensively for its analgesic and antipyretic action. Yet very little is known of its true action.

Its antipyretic action is chiefly by sweating, by cutaneous vaso-dilatation.

The analgesic action is obscure. It is suggested by Sollmann (1948) that the undecomposed acetyl compounds enters the nerve cells more readily.

Aspirin is excreted rapidly in the urine. It lowers the threshold of uric acid and the nitrogeneous compounds and these are excreted in excess.

Sollmann has suggested (1948) that aspirin relieves headache by lowering the intracranial pressure by the mobilization of excess water.

In patients with hypertension who suffer from headache acetyl salicyluric acid may relieve headache by a combination of the following actions.

(1) The antipyretic action produces sweating and generalised cutaneous vaso-dilatation. (It was first thought that aspirin only produced vaso-dilatation in the presence of an elevated temperature, this, however, is not the case. Sollmann states that aspirin even when the temperature is normal produces sweating and vaso dilatation). The sweating will tend to lower the blood volume and thus reduce the venous return to the heart. The cutaneous vaso-dilatation will lower the blood pressure by further reducing the cardiac output. There will thus be a

decrease in the distensile force acting on the cerebral arterioles.

(2) Its analgesic action as Sollmann (1948) states may be due to mobilization of the excess water which is lost in sweat and thus reducing the increased tension. The cushioning effect of the cerebro-spinal fluid will be lost and will allow full dilatation of the arteriole. Thus there will be no sudden dilatation or distension due to sudden drainage of the cerebro-spinal fluid, when the output of the heart increases on rising from bed.

(3) Its action may be potentiated by the delay in excretion due to the damaged kidney. When it is excreted, there will be an increased excretion of the nitrogenous compounds also.

This shows that the full dilatation of the cere - bral arterioles is produced by aspirin; this should further increase the headache. However, although maximum vaso-dilatation is there due to :-

(1) Improved cardiac output.
(2) Lowering of pressure of cerebro-spinal fluid.

The headache as such cannot be appreciated due, one presumes to the analgesic action of aspirin.

This complicated and sometimes paradoxical action of aspirin only gave relief in one patient. It was tried in many patients with frontal and occipital headache in hypertension, and did not give

such dramatic relief as other methods. Presumably where no relief was obtained, the cardiac output was improved and the cerebro-spinal fluid drainage was improved and the distension of the cerebral vessel overcame the analgesic action of the aspirin, resulting in prolongation of the attack of headache.

Palov once stated that in no branch of Science was "conservatism and gradualness" more important than in medicine. A recemt paper by Reid, Watson and Sproull on the action of salicylates would easily explain the relief of headache in these cases. The suggestion in this paper is somewhat revolutionary, maintaining that the administration of salicylates produces an alkalosis and not an acidosis as has hitherto been believed. This alteration therefore, in pH of the plasma to the alkaline side would induce gradual vaso-constriction of the cerebral arterioles and thus relieve the headache.

This paper is highly controversial and the author merely quotes it. It offers an explanation as to the method of relief of headache in these cases, but one must wait and see whether the findings of Reid et al. withstand the test of time.

3. "Head-up" position:

This gave relief in three (60%) out of five cases. Its mode of action is similar when employed in cases of frontal headache.

Elevation of the head of the bed will in health :

(1) Reduce venous return from the limbs.
(2) Lessen cardiac output.
(3) Increase venous drainage from the skull.

In hypertensive patients with cardiac decompensation and renal impairment, sleeping with the head of the bed raised will :

(1) Reduce venous return.
(2) Reduce right-sided cardiac output and thus lessen strain on left ventricle.
(3) Increases venous drainage from the skull.

This increased venous drainage will improve the drainage of cerebro-spinal fluid. The reduced strain on left ventricle will reduce venous pressure generally and here again will improve the drainage of cerebro-spinal fluid.

The emphasis, however in the "head-up" position is more in increased venous drainage from the skull than in the improvement of the cardiac output, although, this does play a part in these cases. The improved cardiac output will increase the blood flow through the kidney, and therefore, the acidosis due to renal impairment and cardiac failure is lessened and only the acidosis from renal impairment affects the vessels.

This represented diagramatically is as follows:-

Diàgram A:-

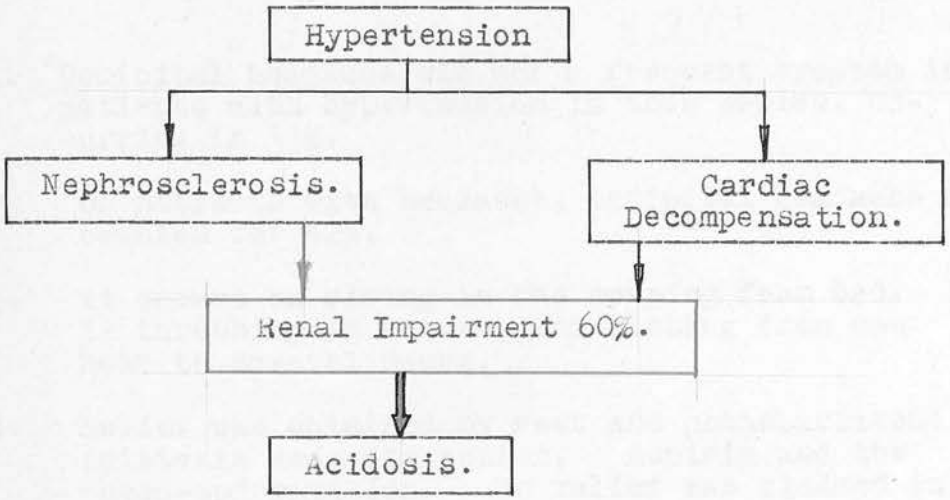
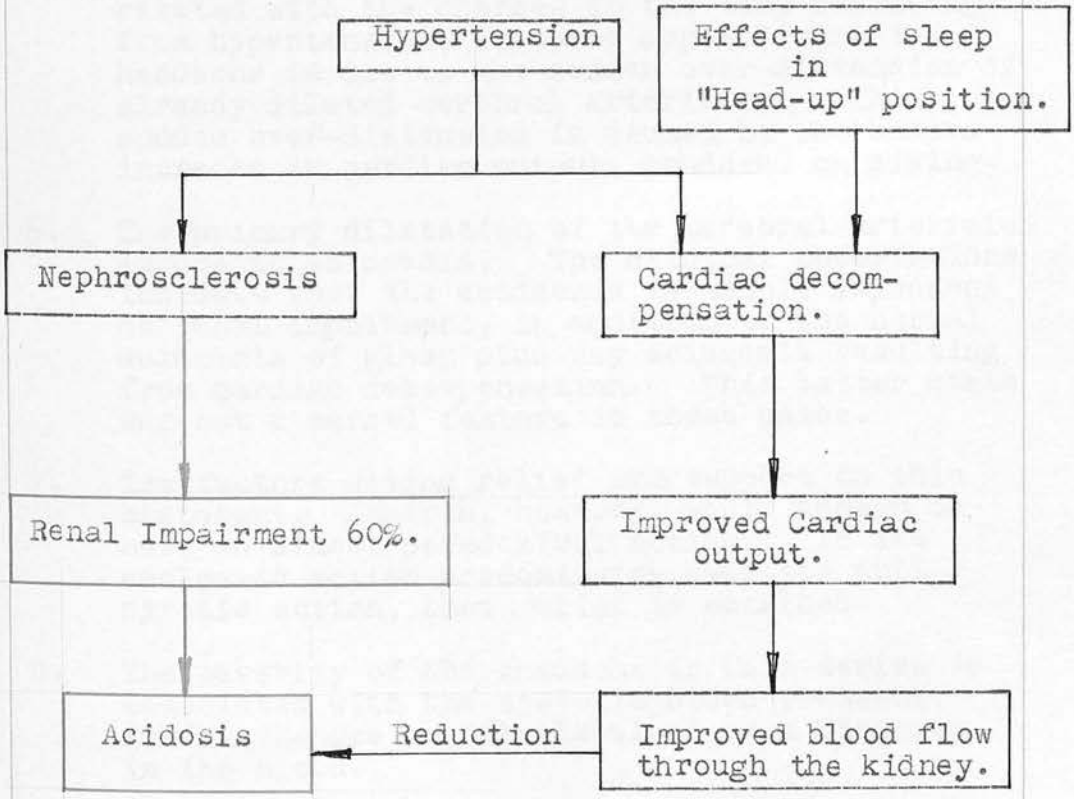


Diagram B:-



Preliminary conclusions based on patients with occipital headache in Essential Hypertension.

1. Occipital headache was not a frequent symptom in patients with hypertension in this series, occurring in 33%.

2. Of patients with headache, occipital headache accounted for 42%.

3. It occurs on rising in the morning from bed. It is throbbing in nature, and lasting from one hour to several hours.

4. Relief was obtained by rest and phenobarbitone, epistaxis and venesection, Aspirin and the "head-up" position. No relief was claimed in one case.

5. When the factors present during sleep, are correlated with the changes in the body resulting from hypertension, it would appear, that the headache is due to the sudden over-distension of already dilated cerebral arterioles. This sudden over-distension is caused by the sudden increase in cardiac output, produced on rising.

6. The primary dilatation of the cerebral arterioles is due to acidaemia. The clinical observations indicate that the acidaemia is mainly dependent on renal impairment, in addition to the normal acidaemia of sleep plus any acidaemia resulting from cardiac decompensation. This latter state was not a marked feature in these cases.

7. The factors giving relief add support to this statement. Aspirin, however, would appear to have an almost paradoxical action. If its analgesic action predominates over its antipyretic action, then relief is obtained.

8. The severity of the headache in this series is associated with the systolic blood pressure, fundal changes and the level of urea nitrogen in the blood.

There are many other types of therapy which workers have used in the relief of headache, such as lumbar puncture, thiocyanates, methonium compound,

rice diet and veriloid. None of these methods were employed in this series.

It is, however, of interest to list the other types of treatment advocated by workers in the treatment of headache of hypertensive origin and compare their results with these in this series.

(1) "Head-up" position: gave relief in 66% of this series.

(2) Rest and phenobarbitone: gave relief in 33% of this series.

(3) Sympathectomy: Gave relief in 8% of this series. If cases are selected, Learmonth claims 90% relief, but these are in selected patients and not in all cases of hypertensive headache.

(4) Venesection: Gave relief in 16% in this series.

(5) Pethidine group of drugs: Gave relief in 8% in this series.

(6) Aspirin: Gave relief in 8% in this series.

(7) Intravenous hypertonic sucrose: Gave relief in a patient with headache who developed hypertensive encephalopathy in our series.

(8) Intravenous Magnesium Sulphate (East): was not tried in this series.

(9) Thiocyanates were not tried in this series. Other authors, however, claim relief.

(10) Vaso-dilators: were not tried in this series. Other authors (East) claim no relief of headache by this means.

(11) Methonium Compounds: were not tried in this series, other workers (Campbell and Robertson) claim good results.

(12) Kempner's Rice diet: Was not tried in this series. The Report of the Medical Research Council claims 70% improvement.

(13) Veriloid: Was not tried in this series.

Patients who experienced no relief from headache:

Two patients in this series experienced no relief from the headache.

One patient had frontal headache and the other patient had occipital headache.

The patient with frontal headache will be considered first :

He was a fisherman aged 53 years. This man had suffered from severe frontal headaches for six months. They were not relieved by rest, phenobarbitone, pethidine or aspirin. The "head-up" position, however, was not tried. The patient's doctor stated that all remedies employed failed to give relief. The patient was admitted to hospital in coma, showing signs of a left-sided hemiplegia. He had Cheyne-Stokes respiration and was deeply unconscious.

The salient features in this patient's examination were a left-sided hemiplegia, Cheyne-Stokes respiration, a blood pressure of $\frac{240}{120}$, fundi Stage IV., enlarged left ventricle, catheter specimen of urine showing sp. gr. 1010, hyaline casts and albumin. A lumbar puncture showed a blood-stained cerebro-spinal fluid at a pressure of 400 mm of H_2O. The patient lapsed deeper into coma and died. The cause of death was cerebral haemorrhage.

The failure to obtain relief would suggest that either another mechanism of headache production was present, or that the methods employed were inadequate.

This patient would correspond to the malignant phase of essential hypertension; where the patient showed fundal changes, renal changes and intractable headache. The diastolic pressure was 120, although not as high as in many cases of this condition, enough criteria were fulfilled to classify this one as the malignant stage of essential hypertension.

It is known that the main pathological feature of the malignant phase is arteriolonecrosis. This condition would so alter the contractile state of the arterioles, that the mechanism of headache production and the methods of relief previously mentioned may no longer apply in this case.

In malignent hypertension there are:

(1) The effects of the gross hypertension on the heart.

(2) The effects of the gross hypertension on the kidney.

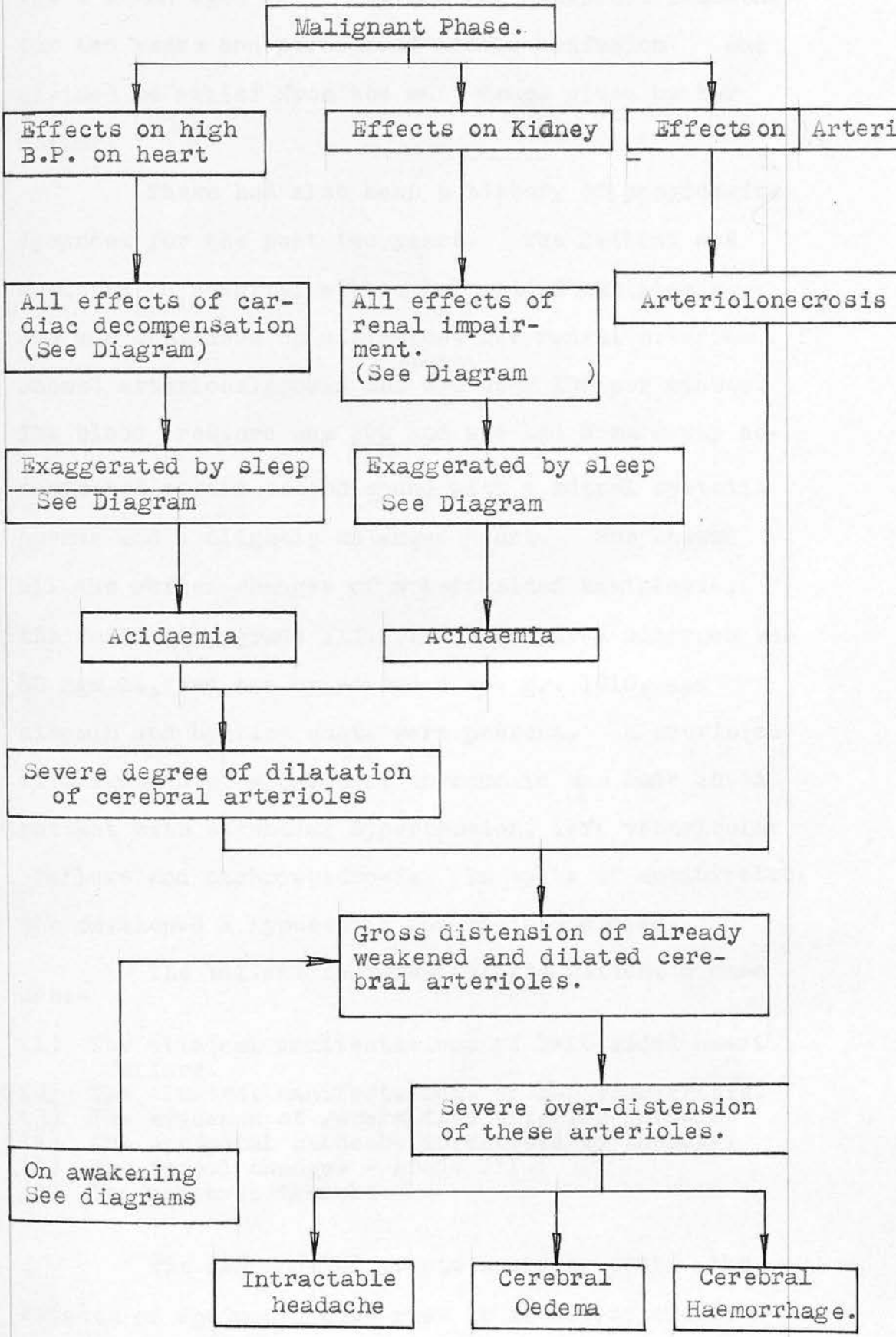
(3) The effects of the gross hypertension on the arterioles generally.

The above leads to the degree of cardiac decompensation, renal impairment and arteriolar necrosis respectively.

The acidaemia from the renal impairment and the acidaemia associated with cardiac decompensation will cause vaso-dilatation. The summation of these effects will be exaggerated during sleep. The resulting severe dilatation of the cerebral arterioles will be even more severe, for these vessels show arteriolar necrotic changes, which thus weakens the wall and allow further dilatation. These greatly dilated arterioles will be subjected to a severe distensile force produced by the sudden impairment in cardiac output on wakening and thus there will be a gross distension of the cerebral arteriole already weakened by necrosis,and overdistended by the increased acidaemia.

This over-distension, it is suggested, leads to intractable headache, (from which this patient suffered) to cerebral oedema with attacks of hypertensive encephalopathy, which was not seen in this case or to cerebral haemorrhage from which the patient died.

These effects can be illustrated diagramatically:-



The other patient,who experienced no relief was a woman aged 72. She had had occipital headache for two years and periods of mental confusion. She claimed no relief from the many drugs given by her doctor.

There had also been a history of progressive dyspnoea for the past two years. The patient was admitted to hospital with a left-sided hemiplegia. She was conscious on admission, her radial arteries showed arteriosclerosis and her pulse was over 100 per minute. The blood pressure was $\frac{200}{110}$ and she had a markedly accentuated aortic second sound with a mitral systolic murmur and a slightly enlarged heart. She showed all the reflex changes of a left-sided hemiplegia, the fundi were grade III., her blood urea nitrogen was 80 mgm.%., and the urine had a sp. gr. 1010, and albumin and hyaline casts were present. A provisional diagnosis of a cerebral thrombosis was made in a patient with essential hypertension, left ventricular failure and nephrosclerosis. In spite of antibiotics, she developed a hypostatic pneumonia and died.

The salient features in this patient'c case are:-

(1) The clinical manifestations of left-sided heart failure.
(2) The clinical manifestations of nephrosclerosis.
(3) The evidence of generalized arteriosclerosis.
(4) The occipital headache unrelieved by therapy.
(5) The fundal changes - grade III.
(6) The cerebral thrombi.

The sequence of events would be that, the effects of age would give rise to arteriosclerotic

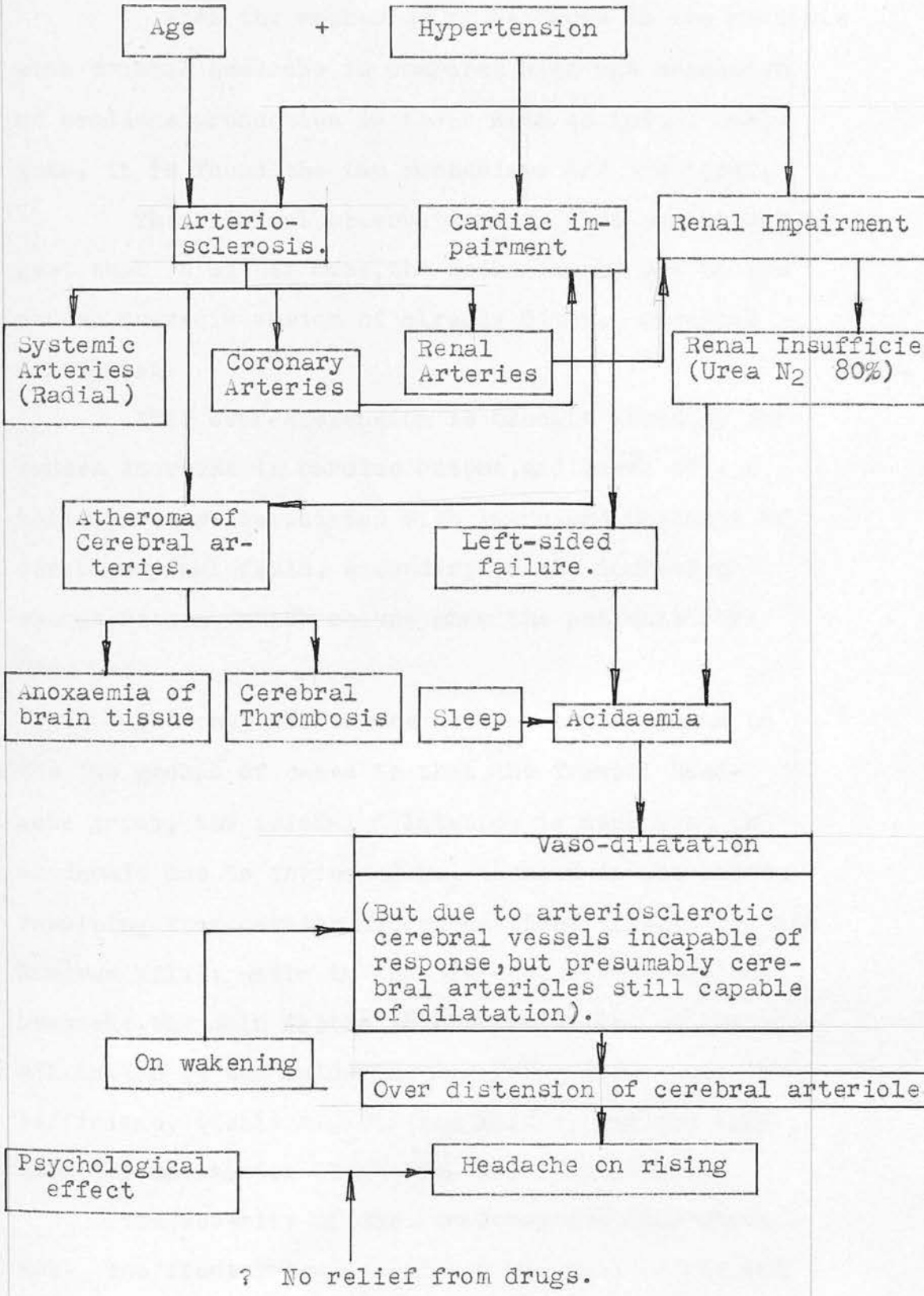
changes which were accelerated by the hypertension. These were seen in the radial arteries,in the cerebral arteries as revealed by the mental confusion, and in the renal arteries as seen by the polyuria, the blood urea nitrogen and the changes in the urine and fundi.

In addition, the hypertension alone did produce left ventricular failure and presumably a further degree of renal impairment.

The acidaemia resulting from the left-sided failure and the acidaemia resulting from the renal failure would produce vaso-dilatation. This is exaggerated as described previously by sleep. The vessels in the brain, which undergo this dilatation are as before, the cerebral arterioles. The main cerebral vessels are the seat of atheroma and are permanently fixed. The headache produced on rising could be explained as before by the over-distension of already dilated cerebral arterioles. It is localized to the occiput as are most cases in this series with a raised blood urea nitrogen. With no permanent changes in the cerebral arterioles, it is surprising, therefore, that this patient claimed no relief from any therapy. This would suggest that either malignant hypertension was present or the patient had a large functional overlay and psychogenic factors played a part. The age, the duration of the illness

(two years) and the fundal changes (grade III.), with a diastolic pressure of only 110, would eliminate the malignant phase of hypertension. She had periods of mental confusion during these two years. This presumably was due to her cerebral arteriosclerosis. It is suggested, therefore, that the cerebral arteriosclerosis with the resulting mental confusion produced a superimposed psychological factor in this patient, and that this was the main factor in her claiming no relief from headache.

Sequence of events:



Discussion on predilection of sites of Headache.

When the mechanism of headache in the patients with frontal headache is compared with the mechanism of headache production in those with occipital headache, it is found the two mechanisms are identical.

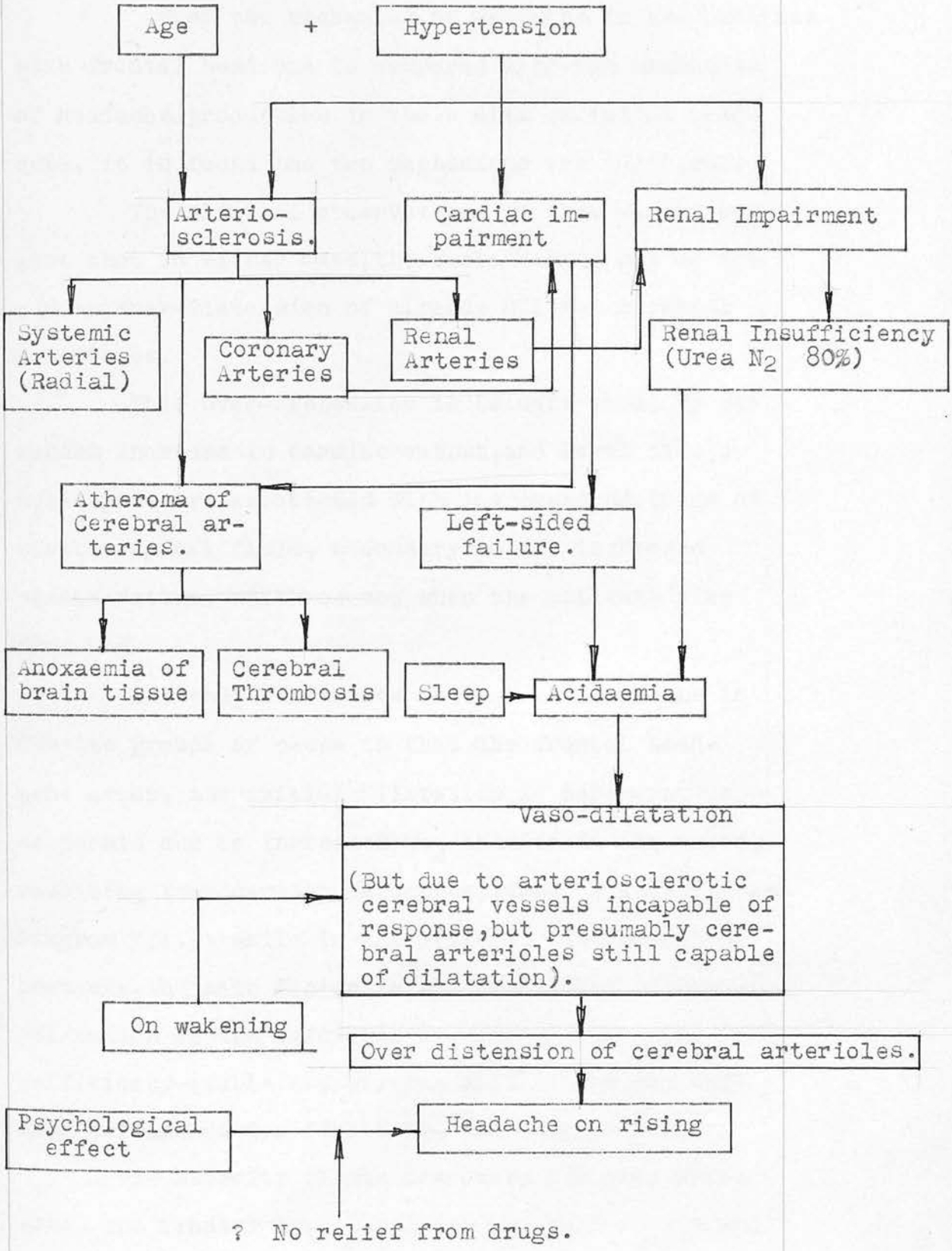
The clinical observations in this series suggest that in either case, the mechanism is due to the sudden over-distension of already dilated cerebral arterioles.

This over-distension is brought about by the sudden increase in cardiac output, and level of systolic pressure associated with increased drainage of cerebro-spinal fluid, secondary to the increased venous return, which occurs when the patients rise from bed.

The only difference between the headache in the two groups of cases is that in the frontal headache group, the initial dilatation is dependent on acidaemia due to increased Co_2 tension in the blood, resulting from cardiac decompensation. (Table VI. and Diagram XII.), while in the patients with occipital headache, the main factor in the production of vasodilatation is the acidaemia resulting from renal insufficiency (Table X., Diagram XXII.), and not cardiac decompensation (Table IX. and Diagram XX.).

The severity of the headaches are also different. The frontal headache group was more severe and the severity was associated with the levels of systolic

Sequence of events:



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The clinical observations in this series suggest that in either case, the mechanism is due to the sudden over-distension of already dilated cerebral arterioles.

This over-distension is brought about by the sudden increase in cardiac output, and level of systolic pressure associated with increased drainage of cerebro-spinal fluid, secondary to the increased venous return, which occurs when the patients rise from bed.

The only difference between the headache in the two groups of cases is that in the frontal headache group, the initial dilatation is dependent on acidaemia due to increased Co_2 tension in the blood, resulting from cardiac decompensation. (Table VI. and Diagram XII.), while in the patients with occipital headache, the main factor in the production of vasodilatation is the acidaemia resulting from renal insufficiency (Table X., Diagram XXII.), and not cardiac decompensation (Table IX. and Diagram XX.).

The severity of the headaches are also different. The frontal headache group was more severe and the severity was associated with the levels of systolic

and diastolic pressure (Diag. VII., VIII. and IX.), fundal changes (Diagram X. and XI), and cardiac decompensation (Table VI.and Diagram XII.)

The occipital headaches were less severe,and the severity was related to the level of systolic pressure. (Diagram XV., XVI., and XVII), fundal changes (Diagram XVIII. and XIX.), and renal insufficiency (Diagram XXI. and XXII.

In other words, the severity of the frontal headache is mainly dependent with the state of the cardiac vascular system, both the heart and the blood-vessels, while the severity of the occipital headache is mainly dependent on the state of the kidneys and to a minor extent to the state of the heart but not the blood vessels.

When the primary mechanism of the headache is the same, why should some be frontal and some be localised to the occiput ?

If all the factors in both types of headache are compared, an explanation for this differentiation may be forthcoming.

The following Table XVII. compares these predisposing and precipitating factors.

Table XVII.

	Frontal	Occipital
(1) Incidence of headache in total cases	47%	33%
(2) Average Age	50 years.	69 years.
(3) Sex-ratio (relation)	6 males : 1 Female	3 males : 2 Females
(4) Average weight	9 st. 12 lbs.	12 st.
(5) Habit:		
Smoking	6 out of 7 - 86%	3 out of 5 - 60%
Alcohol.	2 out of 7 - 29%	3 out of 5 - 60%
(6) Nerve supply of area of headache	Ophthalmic division of the 5th cranial nerve.	(i.) Cervical 1 - 3. (ii.) Lesser Occipital Nerve. (iii) Greater Occipital Nerve.
(7) Acidaemia causing dilatation of cerebral arterioles	(1) Cardiac decompensation (2) Sleep.	(1) Renal impairment. (2) Sleep.
(8) Precipitating factors	Sudden increased cardiac output on rising.	Sudden increased cardiac output on rising.
(9) Relationship of Severity:		
(i) Systolic pressure	R.	R.
(ii) Diastolic pressure	R.	N.
(iii) Fundal changes	R.	R.
(iv) Cardiac decompensation	R.	N.
(v) Blood Urea N_2	N.	R.
(10) Relationship between:		
(i) Blood urea N_2 and B.P.	N.	R.
(ii) B.P. and Fundi	R.	N.
(iii) Fundi and Blood Urea N_2	R.	R.
(11) Relief: "head-up" position	+	+
Rest and Phenobarbitone	+	+
Venesection and Epistaxis	+	+
Aspirin	Not given	+
Pethidine	+	Not given
Intravenous Sucrose	+	Not given
(12) Mortality	14% Cause of death - Cerebral Haemorrhage	20% Cause of death - Uraemia.

Key:- + = Relief. R = Relationship. N = No Relationship.

Comparison of Predisposing and Precipitating Factors:

The factors, Nos. 7, 8, 9, 10 and 11 in Table XVII. have already been considered. The predisposing factors, however, nos. 2-5 in Table XVII. are of interest.

The patients with occipital headache are older than those with frontal headache in this series. This factor is statistically significant (Frontal headache average age 50, occipital headache, average age 69 years of both sexes.

The outstanding difference between the two groups demands some explanation.

The only possible explanations of the phenomenon that occur to the author are:-

(1) Older people experience headache occipitally, while younger people experience headache in the frontal region.

(2) In occipital headache the mechanism is associated with renal impairment, while in frontal headache the mechanism is associated with cardiac decompensation.

(3) The older patients are more prone to cervical osteoarthritis associated with pain in this area. Any mechanism producing headache, therefore, will tend to localize the condition in areas where already the pain threshold is lowered. In this series, however, there was no clinical evidence of such a lesion.

There is no evidence that age plays any part in the localization of pain. The first possibility, therefore, can be discounted.

The nerve supply of the frontal region of the skull is via the ophthalmic branch of the fifth cranial nerve where the nerve supply of the occiput

is via the lesser and greater occipital nerves from Cervical 1 - 3.

The vaso-dilatation and further distension of the cerebral arterioles which occur on wakening in these cases will produce a sudden stretching of the pia mater. This meningeal stimulus could easily be interpreted by the body as arising from other areas supplied by the same nerve i.e. fifth cranial nerve with reference to the pain of the ophthalmic division.

Wolff (1943) has shown that the pain sensitive structures of the head are:

(1) Of the tissue covering the cranium, all were more or less sensitive to pain, the arteries being especially so.

(2) Of the intracranial structures: the great venous sinuses and their venous tributories from the surface of the brain, part of the dura at the base, the dural arteries and the cerebral arteries at the base of the brain, the 5th, 9th and 10th cranial nerves and the upper three cervical nerves were sensitive to pain.

(3) The cranium (including the diploic and emissary veins), the parenchyma of the brain, most of the dura, most of the pia-arachnoid, the ependymal lining of the ventricles and the choroid plexuses were not sensitive to pain.

He further states that stimulation of the pain-sensitive intra-cranial structures on or above the superior surface of the tentorium cerebelli resulted in pain in various regions in front of a line drawn vertically from the ears across the top of the head. The pathways for this pain are contained in the 5th cranial nerve..

Stimulation of the pain-sensitive intracranial structures on or below the inferior surface of the tentorium cerebelli resulted in pain in various regions behind the line just described. The pathways for the pain are contained chiefly in the 9th and 10th cranial nerves and in the upper three cervical nerves.

If his statement is correct, then in the light of our observations, it must appear that patients with frontal headache have the main vaso-dilatation of the cerebral arterioles above the Tentorium cerebelli, while the patients with occipital headache have the main vaso-dilatation of the cerebral arterioles below the tentorium cerebelli. It is difficult to conceive of any such differential dilatation in the cerebral vessels. produced by acidaemia. Nevertheless, a differential degree of vaso-dilatation would be a satisfactory explanation of this localization of headache in this series.

This suggestion supports the work of Norcross who investigated the blood flow in the brain. He studied cerebral blood flow through the parietal area of the cat's brain using the thermolbetric method of Gibbs. He found that the inhalation of Co_2 caused a tremendous increase in blood flow, increase being proportional to the percentage of gas in the mixture inhaled. Oxygen on the other hand resulted in a diminution in the blood flow through this area.

Furthermore, the experiments of Jacobi, Schmidt and others on cerebral circulation indicate that the

alteration of the PH of the blood from whatever cause would affect the size of the minute vessels of the brain.

Norcross also stated that "May it not be that a difference in the site of the dilatation in these cases accounts for the difference in the response"?

Schmidt has contributed an important paper on this subject. He has shown that the reaction to the same stimulus is not necessarily the same in different parts of the brain. Or in other words, the increased blood flow and vaso-dilatation resulting from acidaemia or excess Co_2 content of the blood may not be generalized, but only affect certain portions of the brain.

It would appear, therefore, in the patients in this series, that for some reason, when the acidaemia is mainly due to cardiac decompensation, resulting in excess Co_2 retention, the headache is situated frontally.

Yet, when the acidaemia results from kidney impairment, the headache is situated occipitally.

In the first instance, the dilatating agent is Co_2, and in the second case, the dilating agent is the acidaemia of retained waste products of metabolism plus possibly the excess Co_2, resulting from cardiac decompensation, if present.

Another explanation would be, that the acid-aemia, whatever the cause, produces the vaso-dilatation, and that the pain from this, is experienced, in areas of the scalp where the pain threshold is already lowered by pre-existing or concommitant disease, such as cervical osteoarthritis or fibrositis.

Yet another explanation for the headache, be it occipital or frontal is psychological. The older patients with more change of cervical osteoarthritis and realizing they have hypertension may refer any sensations they experience to the occiput or cervical region, and state they have a headache.

Worry, emotional stresses and strains plus the anxiety of the knowledge that hypertension is present, may result in patients, who experience any sensation in the frontal region, perhaps due to spasm of the frontal muscle, stating they have a headache in this region.

Furthermore, constant stroking the front or back of the head may in time induce a sensation of headache in such individuals.

In any case of essential hypertension, the psychological elemant cannot entirely be ruled out.

In conclusion, I would submit to leave this subject open at present, pending the accumulation of more information with respect to differential dilatation of the intra-cranial vessels.

Habits:

In this series of cases, it is found that there is a difference in the habits of the patients with occipital and with frontal headache in respect of smoking and drinking.

In patients with hypertension whose headache is localized to the frontal area 86% had a history of smoking and only 29% were alcoholics. In patients with hypertension whose headache was localised to the occipital region 60% gave a history of smoking, and 60% gave a history of alcoholism. These figures are statistically and should be clinically significant.

In an endeavour to explain this difference in habits one must mention briefly the effect of (a) Tobacco and (b) alcohol on the body.

Tobacco, when smoked produces many factors, which may be absorbed into the body. The most important are nicotine, carbon-monoxide, certain pyridins and certain volatile hydrocarbons. The result effects of these on the body are well known - there is local irritation of the pharynx, larynx and bronchial tree, there is the irritant effect on the myocardium producing extrasystoles; there is the irritant effect on the arteries and arterioles producing vaso-constriction and there is the effect on the central nervous system acting as a general and autonomic stimulant.

The only factors, if any, which are likely to be of importance in predisposing to frontal headache in hypertension, are possibly the effects on the blood vessels and heart. It is difficult to imagine how the production of extrasystoles would play any part, therefore, the effect on the blood vessels may be a factor.

Vaso-constriction is the general effect of smoking. This may affect:

(1) The blood vessels of the coronary system.
(2) The blood vessels of the eye.
(3) The cerebral vessels of the brain.

(1) The Coronary Vessels: Constriction of these vessels or any permanent change which may result from excessive smoking will impair the heart's action, and tend to lower its efficiency. This will augment any coronary insufficient present already resulting from the hypertension. The factor, therefore, of cardiac embarrassment in the mechanism of frontal headache production will be increased.

(2) The Vessels of the eye: Heavy smoking results in tobacco amblyopia. Habitual smokers, however, do not show any occular change. The factor, therefore, of eye strain, with resulting spasm of the frontal muscle, although a likely possibility, would appear to play no part

3. Constriction of the cerebral vessels produced by smoking should, indeed reduce the severity and lessen the incidence of headache, provided the individual was smoking on the occasion that headache was present.

Smoking, therefore, if it has any bearing clinically, as statistically it should have, would appear to act by increasing the cardiac impairment by no other obvious manner.

The effects of alcohol on the body are well known - it is a sedative, a vaso-dilator, initially a stimulus to gastric secretion and of course, a central nervous system irritant and later depressant.

Of these actions, its action as a vaso-dilator would appear to be important. Alcohol acts as a generalized vaso-dilator, therefore, coronary, skin and cerebral vessels undergo this dilatation. The cardiac efficiency should therefore, be increased, the cutaneous vaso-dilatation should increase sweating, reduce the blood volume and tend to lower the venous pressure. The cerebral vaso-dilatation,will naturally add to the dilatation resulting from acidaemia present due to cardiac and renal inefficiency.

It can be seen, therefore, that alcohol would tend to increase the severity of the headache and increase its incidence in patients with hypertension who suffer from headache. 60% of the patients with occipital headache were alcoholics. Alcohol, however, will only add to the factors producing headache,where it is actually in the blood stream. It can play no part in the headache production, when the patient has not drunk alcohol, so that all one can say in respect of the alcohol as a factor in headache production is

that when drunk, it will add to the factors producing headache, but a history of alcoholism cannot as far as it can be seen influence the site of the headache. Tobacco and alcohol, therefore, although influencing the severity of the headache cannot per se play any part in the localization of the headache.

Both factors therefore, although statistically significant remain as unexplained predisposing factors in the localization of headache.

Weight: It can be seen in this series that patients with occipital headache in essential hypertension are definitely heavier than are those who experience frontal headache. The average difference in weight between the two groups amounts to 2 stones 2 lbs. Here again this factor may be significant and may merely indicate that the older age group (average age 69) with occipital headache are heavier than the younger age group (average age 50 years) who complain of frontal headache.

Before any final observations are made on this subject the cases of chronic glomerulo-nephritis with resulting hypertension and headache must be briefly reviewed.

Prognosis and Conclusions of Headache in Essential Hypertension:

Clinical observations and analysis of patients with essential hypertension, who suffer from headache has shown that :

(1) The headache is of two types (a) frontal and (b) occipital.

(2) Frontal headache occurred in 58% of these patients with headache and in 47% of the total group of patients, with and without headache.

(3) Occipital headache was less frequent. It occurred in 41% of patients with headache but in only 33% of the total group.

(4) 75% of the patients with essential diastolic hypertension, therefore, suffered from headache. This figure is comparable with the finding of Keith et al., who showed an incidence of headache in 78% of their cases of diastolic hypertension who were surgically treated. Like the findings of Keith, our results are considerably higher than the figures given by Bechgaard who had only an incidence of 23%.

(5) The character of the headache was the same in both groups, it occurred on wakening or rising from bed in the morning and was throbbing in nature.

(6) The duration of the attack was from half an hour to several hours but tended to last longer when it was situated occipitally.

(7) Relief could be obtained by many factors - epistaxis, sympathectomy, pethidine, rest and phenobarbitone, intravenous hypertonic sucrose and sleeping in the "head-up" position in the group with frontal headache. Rest and phenobarbitone, epistaxis and venesection, aspirin, and again sleeping in the "head-up" position gave relief to patients with occipital headache.

No relief could be obtained in one patient with frontal headache and in one patient with occipital headache.

(8) In both groups of cases, our investigations have shown that the precipitating factors in the production of frontal or occipital headache is the sudden over-distension of dilated cerebral arterioles. This over-distension results from a sudden increase in cardiac output and level of blood pressure produced by rising or wakening.

Observations on these patients and their response to the many therapeutic measures used, show that a gradual increase in the cardiac output with a resulting gradual elevation of the blood pressure does not produce headache.

(9) The cerebral arterioles are dilated initially during sleep as a result of acidaemia. In the patients with frontal headache, the acidaemia results from the effects of sleep but mainly from cardiac decompensation with resulting excess Co_2 retention.

In the patients with occipital headache, the acidaemia results partly from sleep but mainly from the acidosis of renal impairment; cardiac factors seem to play little or no part in the headache production in the occipital group.

(10) The site of the headache and the severity of the headache in this series indicate that when the headache is frontal then there is cardiac decompensation and the severity is a direct indication of the degree of cardiac efficiency.

When the headache is situated occipitally then there is renal impairment and the severity of the headache is dependent upon the degree of renal impairment.

(11) Comparison of the many factors present in both groups of cases show that patients with occipital headache are both older and heavier than patients with frontal headache. Patients with frontal headache smoke more heavily but drink less alcohol than do patients with occipital headache. These factors, however, seem to play no part in the etiology of the headache.

(12) The explanation of the different sites of the headache in these patients, is a differential degree of vaso-dilatation of the cerebral arterioles. In frontal headache, the dilatation is due to acidaemia of Co_2 retention and affects the vessels above the tentorium cerebelli and is thus felt in the distribution of the ophthalmic division of the fifth cranial nerve.

In occipital headache, the dilatation is due to the acidaemia of renal impairment caused by the waste products of metabolism and affects the vessels below the tentorium cerebelli and the pain is thus felt in the distribution of first, second and third cervical nerves.

Another explanation would be, that the acid-aemia, whatever the cause, produces the vaso-dilata-tion and that the pain from this is experienced in areas of the scalp where the pain threshold is already lowered by pre-existing or concommitant disease such as cervical osteo-arthritis or fibrositis. In this series, however there was no clinical evidence of such a lesion.

(13) The mortality was 14% in the patients with frontal headache, death resulting from a cardio-vascular catastrophy. In occipital headache, the mortality was 20%, death resulting from uraemia.

2. Chronic Glomerulo-Nephritis.

Hypertension is a common feature in patients who suffer from chronic glomerulo-nephritis. The diseased kidney, gradually becoming fibrotic and ischaemic produces an excess quantity of renin. (Goldblatt). There is, as a result of this an increase in the peripheral resistance of the blood vessels and hypertension results. In time, permanent changes will occur in the blood vessels and heart. A patient therefore who is suffering from chronic glomerulo-nephritis will show all the effects of renal impairment together with the effects of hypertension.

The cases of Chronic glomerulo-nephritis which are described fully in the appendix are analysed as follows:- p. 178

Key to Table

+	=	Yes.
F	=	Frontal.
O	=	Occipital.
T	=	Throbbing.
M	=	Moderate.
S	=	Severe.
E	=	Enlargement.
N.C.E.	=	No clinical enlargement.
L.A.D.	=	Left axis deviation.
A	=	Admission.
D	=	Discharge.
M.S.M.	=	Mitral Systolic Murmur.
A_2 ++	=	Second Aortic sound accentuated.
R	=	Relief.

Analysis of Cases of Chronic Glomerulo Nephritis.

Table XVIII.

Sex	Male	Female	Male	Female	Male
Age	45	45	52	64	68
Weight	8 st.8 lbs.	10 st.	-	14 st.	8 st. 6 lbs.
Habit:					
Smoking	+	-	+	-	+
Alcoholic	+	-	+	-	-
Family History	Not relevant	Mother - Hemiplegia	Mother - Hemiplagia	Not relevant	Not relevant
Previous Illness	Acute Nephritis - 6 yrs.	Not relevant	Not relevant	Scarlet Fever at 48	Not relevant
Presenting Symptons.	Poor Eyesight, Insomnia - 4 days, Nocturnal frequency - 6 yrs.	Dyspnoea - 3 days	Diarrhoea Anorexia and vomiting - 2½ months	Dyspnoea - 5 years. Swollen face and feet - 6 weeks	Dyspnoea - 3 weeks. Swollen face - 6 months.
Headache:	+	+	+	+	+
Duration	1 week	3 weeks	1 week	5 years	6 months
Site	F	F and O	F	F	F
Character	T	T	T	T	T
Onset	Anytime and in morning	Anytime and in morning	Constant, but worse in morning	in morning	in morning
Severity	M	M	S	M	M
Precip. factors	Rising from bed	Rising from bed	Rising from bed	House-work	On wakening
Ass. Symptoms	Nausea	Failure to concentrate	Nausea	-	Nausea
Duration of Attack	Up to ½ hour	Several hours	Several hours	More than 2 hours	About 1 hour
Relief	+ Rest and diet	Slight By Aspirin	No relief	+ Rest and diet	+ Rest and diet
B.P.	A D				
Systolic	160 150	180 150	170	160 150	180 160
Diastolic	100 90	100 90	100	100 90	100 110
Heart	N.C.E.	E	E	N.C.E.	E
E.C.G.	-	L.A.D + Extra Systoles	-	-	L.A.D.
Basal Crepitation	-	Few	Few	-	Few
Fundus oculi	III.	IV.	IV.	I.	III.
Blood Urea Nitrogen	A. 82 mgm% D. 54 mgm%	63 mgm% 32 mgm%	116 mgm%	118 mgm% 21 mgm%	50 mgm% 30 mgm%
Urea-Range	Sp.gr. Gm/100c.c.				
Con.	1018 - 3	1010 - 1.5	-	1018 - 2	1016 - 2·4
dil.	1002 - 1	1008 - 1.2		1012 - 1·2	1010 - 1·2
Co_2 Combining power	54 Vols %	35 Vols.%-50 Vols.%	40 Vols.%	44 Vols.%	52 Vols. %
Urinary Vols. in 24 hrs in c.c.	1500	1500	1000	1200	800
Reaction	A	A	A	A	A
Sp. gr.	1016	1018	1010	1016	1020
Albumin	+	+ +	+	+	+
Blood	+	+ +	-	-	+
Sugar	-	-	-	-	-
Casts	Granular and Hyaline	Granular and Hyaline	Granular and Hyaline	Granular and Hyaline	Granular
Treatment	Rest, Restricted Salt and low protein diet	Rest, O_2 therapy Aminophylline and dietetic	Rest and Symptomatic	Rest, salt free diet. Restricted protein etc.	Rest and low protein diet
Result	R	R	Died	R	R

Table XVIII[A].

Sex	Male	Male	Male	Female	Male
Age	16	38	39	53	46
Weight	9 st. 12 lbs.	9 st. 7 lbs.	11 st. 5 lbs.	-	9 st. 3 lbs.
Habit: Smoking	-	+	-	-	+
Alcoholic	-	+	-	-	+
Family History	Not relevant	Not relevant	Not relevant	Not relevant	Not relevant
Previous Illness	Scarlet Fever 7 years ago	Acute Nephritis 17 years ago	Not relevant	Not available	Scarlet Fever 6 years ago.
Presenting Symptoms.	None for routine examination	Lethargy - 1 month Poor vision - 4 days Nocturnal Frequency 4 months	Lethargy - 3 months Swollen face in morning - 1 week Nocturnal Frequency 6 months	Drowsiness - 6 weeks	Dizziness - 6 weeks Nausea - 1 week Nocturnal Frequency 1 year.
Headache	-	+	+	+	+
Duration	-	1 month	3 months	6 weeks	6 weeks
Site	-	O	F	F	F
Character	-	T	T	T	T
Onset	-	in morning	in morning	Constant but worse in morning	in morning
Severity	-	S	S	S	M
Precip. Factors	-	On awakening	on rising	House-work and on rising	On rising
Ass. Symptoms	-	Nausea	Nausea	Nausea	Dizziness and Nausea
Duration of attack	-	1 - 2 hours	Several hours	Several hours	About 1 hour
Relief	-	No relief By Sod.Amytal and aspirin	No relief By Sodium Amytal and Aspirin	No relief	+ Rest and diet
Blood Pressure: Systolic / Diastolic	150 / 95 — 150 / 95	200 / 140	240 / 140	200 / 150	160 / 100 — 140 / 90
Heart	N.C.E.	N.C.E. M.S.M. A_2 ++	E A_2 ++	E Pericardial friction rub	E A_2 ++
E.C.G.	-	L.A.D.	-	-	L.A.D.
Basal Crepitation	-	few	few	few	-
Fundus Oculi	Normal	IV.	IV.	IV.	III.
Blood Urea Nitrogen	23 mgm.%	155 mgm.%	121 mgm. %	200 mgm.%	50 mgm.%, 30 mgm.%
Urea Range Con.	Van Slyke 66%	-	1010 - 1	-	1016 - 2·5
Urea Range Dil.			1010 - 0·5		1010 - 1·2
Co_2 Combining power	-	24 Vols. %	48 Vols.%	-	48 Vols.%
Urinary Vol in 24 hrs in c.c.	1500	3000	2000	1500	2500
Reaction	A	A	A	A	A
sp. gr.	1012	1010	1010	1013	1010
Albumin	Trace	++	+	+	+
Blood	-	+++	-	+	-
Sugar	-	-	-	-	-
Casts	Occasionally Granular	Granular and blood casts	Granular and Hyaline	Granular	Granular and Hyaline
Treatment	Nil	Rest and Symptomatic	Rest and Symptomatic	Rest and Symptomatic	Rest and low protein diet.
Result	-	Died	Died	Died	R.

Comparison of Patients with Headache and without Headache in Chronic Glomerulo-Nephritis.

Table XIX.

	With Headache	No Headache
Number	9	1
Males	6	1
Females	3	-
Average age	50 years	16 years
" Weight	10 st. 2 lbs.	9 st. 12 lbs.
Habit:		
Smoking	5	-
Alcoholic	4	-
Average age:		
(a) Males	48 years	16 years
(b) Females	54 years	-
Average weight:		
(a) Males	9 st. 6 lbs.	9 st. 12 lbs.
(b) Females	12 st.	-
Family History of Hypertension :		
(a) Males	1	-
(b) Females	1	-
Previous illness*		
(a) Acute Nephritis	2	-
(b) Scarlet Fever	2	1
(c) Tonsilitis	-	-
Average B.P.	Admission / Discharge / Lability	Admission / Discharge / Lability
(a) Systolic	183 / 173 / 10	150 / 150 / Nil
(b) Diastolic	114 / 111 / 3	95 / 95
Heart	5 clinically enlarged A_2 ++ in 3 cases	Normal
E.C.G.	Left Axis deviation in 4. Not taken in 5	Not taken
Basal crepitation	6	-
Fundus Oculi	Grade I. in 1 case; " II. in 0 "; " III. in 3 "; " IV. in 5 "	Normal
Average Blood Urea Nitrogen	Admission 106 mgm.% Discharge 33 mgm.% out of 5	Admission 23 mgm.% Discharge 23 mgm.%
Urea Range:	sp.gr. Gm/100 c.c.	
Con.	1014 - 2·06	Van Slyke - 66 % Normal
Dil.	1008 - 1·05	
Average Co_2 Combining power	43 vols.%	Not done.
Urine (Average)		
(1) Volume	1666 c.c.	1500
(2) sp. gr.	1013	1012
(3) Albumin	9 cases	Trace
(4) Blood	5 cases	-
(5) Casts	Granular and Hyaline in all	Occasionally Hyaline.
Number of deaths	4	-

Results of Tables XVIII. & XIX.

(1) Incidence:

Of ten patients with chronic glomerulo-nephritis:
9 patients suffered from headache - 90%
1 patient had no headache - 10%

The headache was frontal in 8 cases and occipital in two cases.

(2) Sex-incidence:

In the group with headache, there were six males and three females, a ratio of 2M : 1F.

(3) Age-incidence:

There is no significance in the age distribution of these cases. Like the patients with essential hypertension, the females tended to be older than the males. The average age of the females was 54 and the males 48 years.

(4) Habit:

50% had smoking habit.
40% were alcoholic.

(5) Family History:

There was no significant evidence of family history playing a part in these cases except that in two patients with headache (1 male and 1 female) there was a family history of hypertension.

(6) Previous Illness:

The previous history of the individuals showed that two of the patients with headache had had acute nephritis and two of these patients had had scarlet fever. In the patient with no headache, there was a previous history of scarlet fever.

(7) Blood Pressure:

The blood pressure levels - both systolic and diastolic were higher in the patients with headache.

(8) Heart :

In the patients with headache, there was evidence of clinical enlargement of the heart in five cases and electrocardiographic evidence in four cases.

(9) Urine Examination:

The patients with headache showed marked urinary changes and five of these cases also had blood in the urine.

(10) Blood-Urea-Nitrogen:

The blood urea nitrogen on admission in the nine patients with headache averaged 106 mgm.%. In five patients who were discharged from hospital, the average blood urea nitrogen was 33 mgm.%. In the single case with no headache the urea nitrogen remained at 23 mgm.%.

(11) Urea Range:

The Urea-range was performed in all but two patients. This showed gross renal impairment. In the one patient without headache a Van Slyke urea clearance was performed and this showed a value of 66% of the normal, which is not grossly impaired.

12. The Co_2 Combining power of the blood:

The Co_2 combining power showed evidence of acidaemia in the eight cases in whom it was performed. The average was 43 vols.%.

(13) Retinal changes:

The fundal changes were present in all the patients with headache. They were as follows:-

Grade	I.	1 case.
Grade	II.	-
Grade	III.	3 "
Grade	IV.	5 "

The patient with no headache had a normal fundus.

It can be seen from Tables XVIII & XIX that the headache was either frontal or occipital.

Table III. compares the clinical features of these two groups.

Comparison of Frontal and Occipital Headache in Chronic Glomerulo Nephritis.

Table XX.

	Frontal	Occipital
Number	(a) Total 8 out of 10 (b) Headache 8 out of 9	(a) Total 2 out of 10 (b) Headache 2 out of 9.
Sex	5 M. : 3 F.	1 M. : 1 F.
Average age	52	42
Average weight	10 st. 4 lbs.	9 st. 10 lbs.
Average age: (1) Males (2) Females	50 54	38 45
Average weight (1) Males (2) Females	9 st. 6 lbs. 12 st	9 st. 7 lbs. 10 st.
Habit: Smoking Alcoholic	4 3	1 1
History of Headache Time of onset	1 week - 5 years in morning 0 Any time 2 Constant 2	3 weeks - 4 weeks in morning 1 Any time 1
Severity (1) Mild (2) Moderate (3) Severe	- 5 3	- 1 1
Duration Character Relief	½ hour - several hours Throbbing Relief 4 Slight " 1 No " 3	2 hours - several hours Throbbing Slight relief 1 No relief 1
Average B.P. (1) Admission (2) Discharge	181/111 170/108	190/120 175/115
Average B.P. Severity Mild " Moderate " Severe	- 168/100 203/130	- 180/100 200/140
Fundal changes: Severity Mild " Moderate " Severe	8 - III in three, IV in one, I in one. IV in three	2 - IV in one. IV in one.
Cardiac efficiency: Severity Mild " Moderate " Severe	- Normal in 2, I_B in two, II^A in one II^A in one, II^B in one & III in one	- I in one. II^B in one.
Blood Urea Nitrogen Severity Mild " Moderate " Severe	- 72 mgm.% 146 mgm.%	- 63 mgm.% 155 mgm.%
Average Co_2 Combining power Severity Moderate " Severe	46 Vols. % 49 Vols. % 44 Vols. %	30 Vols. % 35 Vols. % 24 Vols. %
Urinary findings: " Mild Severity Moderate (Output (sp. gr. (Albumin (Casts	- 1500 c.c. 1012 + Granular and Hyaline in 3	- 1500 c.c. 1018 + +
Severe (Output (sp. gr. (alb. (R.B.C. (Casts	1500 1011 + + +	3000 c.c. 1010 ++ +++ ++
Mortality Severity Mild " Moderate " Severe	- - 3	- - 1

Preliminary conclusions from Tables XVIII., XIX & XX.

The analysis of these patients with chronic glumerulo nephritis show that:-

(1) Incidence of headache:

90% of the patients with chronic glomerulo-nephritis had headache. This is statistically significant.

(2) Sex Incidence of patients with headache :

Of these cases with headache, 66% were males and 33% females.

(3) Age incidence of patients with headache:

The average age of the male patients was 48 years, and the females 54. Of the patients with frontal headache, the average age was 50 years for males and 54 years for females. In the group with occipital headache the age for men was 38 and women 45 years.

It can be seen, therefore, that in either type of headache, women are older than men. Occipital headache, however, occurs in a younger age group both in men and women than in the case of frontal headache.

(4) Average weight of patients with headache:

In the patients with frontal headache, the average of the males was 9 stones, 6 lbs. and the women 12 stones. In the group with occipital headache, the average weight of the men was 9 stones 7 lbs. and of the women 10 stones.

These figures show no significant difference in the two groups suffering from headache but they do show that the average weight of the woman in both groups is greater than the average weight of the men

(5) Habits:

50% had smoking habit.
40% were alcoholics.

(6) Family History of patients with headache:

There is no family history of nephritis in these cases. Two patients, however, gave a family history of hypertension. This, however, is not significant.

(7) Previous illness of patients with headache:

Four patients with headache had a pre-existing history; two patients had acute nephritis and two patients had scarlet fever. This is significant.

(8) Characteristics and precipitating factors of the headache:

(a) The occurrence of the headache in these cases was, 70% experienced it on rising in the morning, 20% experienced headache at any time and in 20% the headache was constant.

(b) Frontal headache was more common than the occipital.

(c) The character was the same in every case, being throbbing in nature.

(d) The duration of the attack varied from half an hour to several hours in the cases of the frontal headache, and from two hours to several hours in the cases with the occipital headache.

(e) The severity of the headache was different in the two groups. In patients with frontal headache, five cases had a moderate headache and three were severe. In the occipital headache group, one was moderate and one severe.

(f) No relief was obtained in 40% of the cases.

(9) Lability of the blood pressure:

In patients with headache in this series, the blood pressure levels fell while under treatment. The average fall of the systolic pressure was 10 mm Hg. and of diastolic 3 mm Hg. These figures are hardly significant.

(10) Cardiac Efficiency:

The cardiac efficiency in patients with frontal headache was as follows :

Normal	-	2 cases.
Grade I.	-	2 cases.
" IIA	-	2 cases
" IIB	-	1 case
" III	-	1 case.

in
While/the patients with occipital headache, the cardiac efficiency was Grade I. in one case and grade IIB in another.

(11) Renal functions:

100% of these patients showed evidence of renal impairment. The degree of renal impairment as told by the urinary volume, specific gravity, blood urea nitrogen, Co_2 combining power and the presence of blood in the urine indicates that occipital headache shows a greater degree of renal impairment than does frontal headache.

(12) Retinal changes:

The retinal changes were more pronounced in the occipital group. In the two patients with occipital headache, both had grade IV. i.e. 100%. In the group with frontal headache one had grade I. i.e. 13%; three had grade III. 37% and four had grade IV. - 50%.

This is seen more clearly:

Grade	Frontal	Occipital
I.	13%	-
II.	-	-
III.	37%	-
IV.	50%	100%

(13) Co_2 Combining power of the blood:

The Co_2 combining power was estimated in eight of the nine patients with headache. In the patients with frontal headache, the average Co_2 combining power was 46 Vols.%., while the Co_2 combining power in the patients with occipital headache was 30 vols.%. This fact is significant.

(14) Mortality:

One patient out of two with occipital headache died - 50%.; while three out of eight with frontal headache died - 37%.

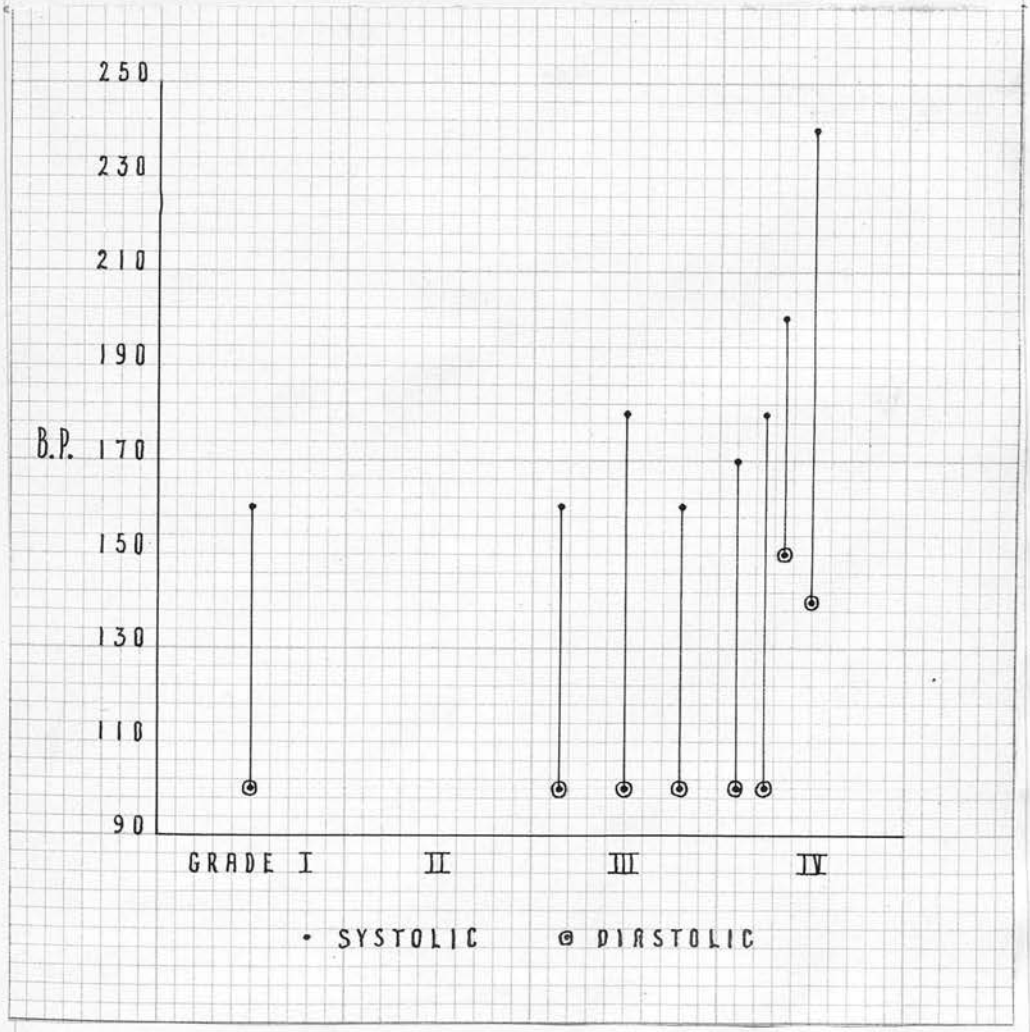
The patients with occipital headache are too small to compare with frontal headache but renal, fundal and mortality observations indicate that occipital headache has the worst prognosis and is associated with more renal impairment in this series.

Relationship of Pathological and Biochemical Changes with the clinical features of the headache in Chronic Glomerulo-Nephritis.

A. FRONTAL HEADACHE.

(1) B.P. levels and fundal changes:

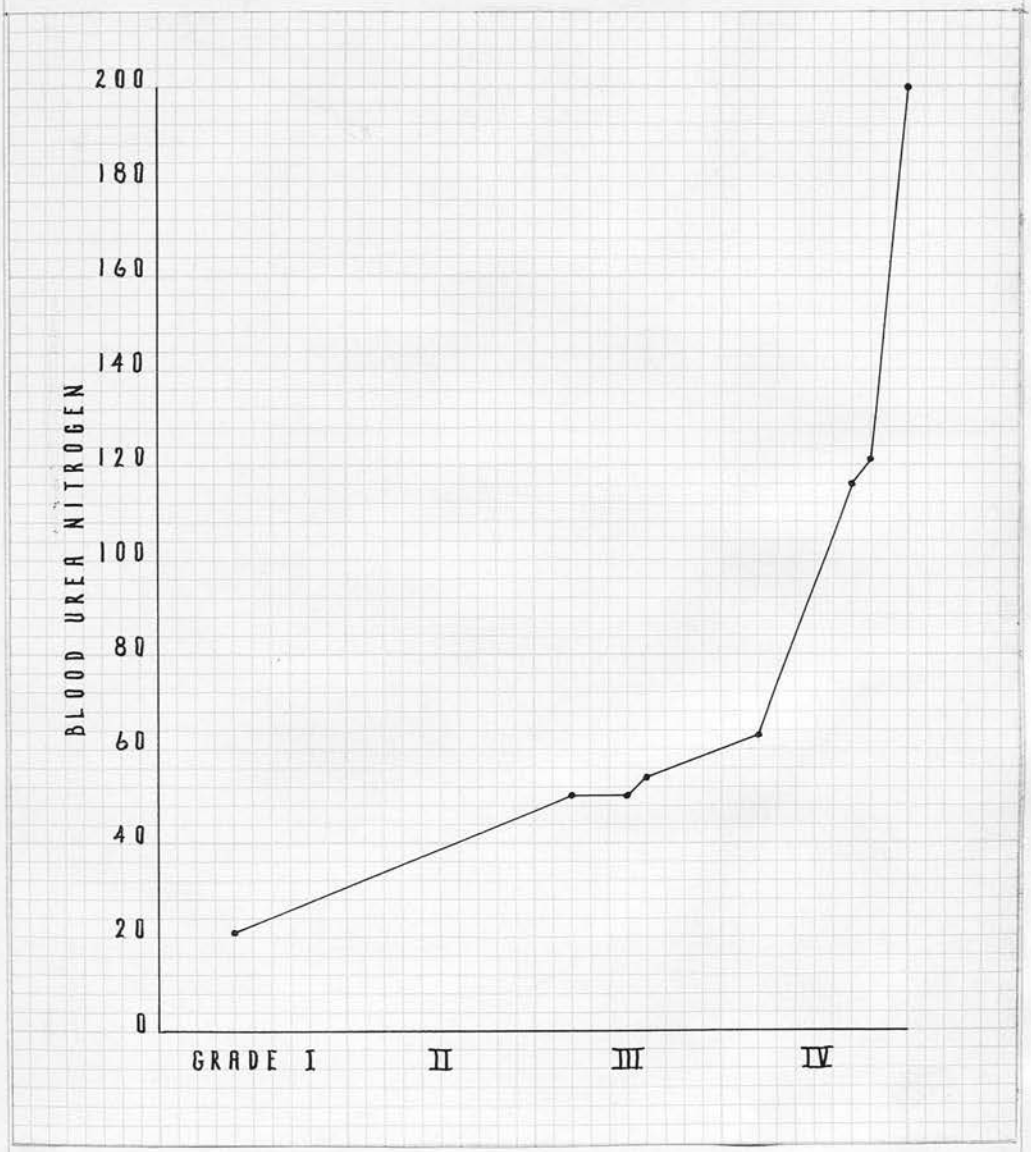
The relationship between the levels of systolic and diastolic pressure and the fundal changes in these patients with frontal headache is seen in the following diagram. XXVIII.



This suggests but does not show definitely a correlation between the blood pressure levels and the fundal changes.

(2)Fundal Changes and blood Urea Nitrogen.

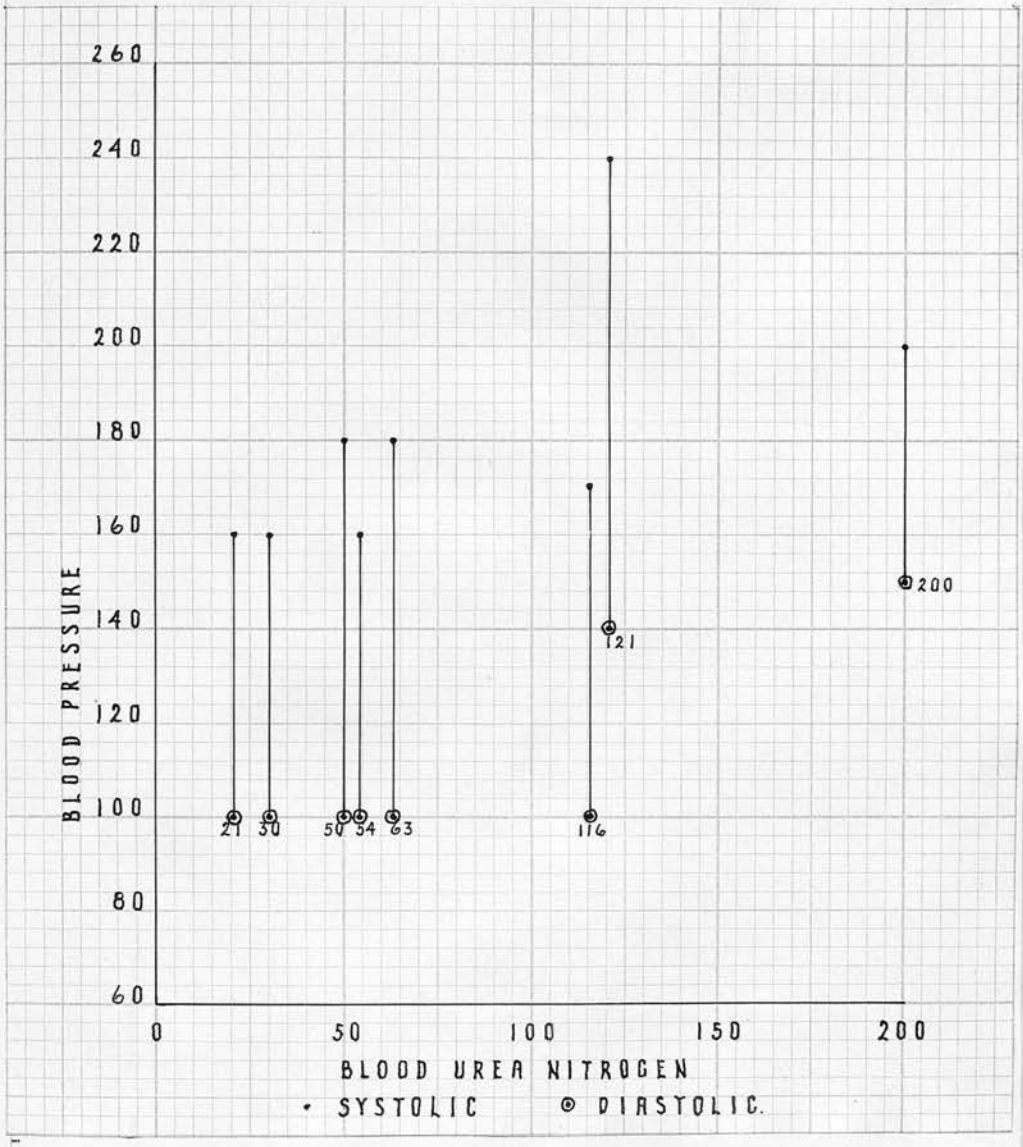
Diagram XXIX illustrates the relationship between the fundal changes and the levels of blood urea nitrogen.



There is a definite correlation between the blood Urea Nitrogen and fundal changes, in patients with frontal headache in Chronic Glomerulo-Nephritis.

(3) B.P. levels and Blood Urea Nitrogen:

Diagram XXX shows the blood pressure levels with the corresponding levels of blood urea nitrogen.

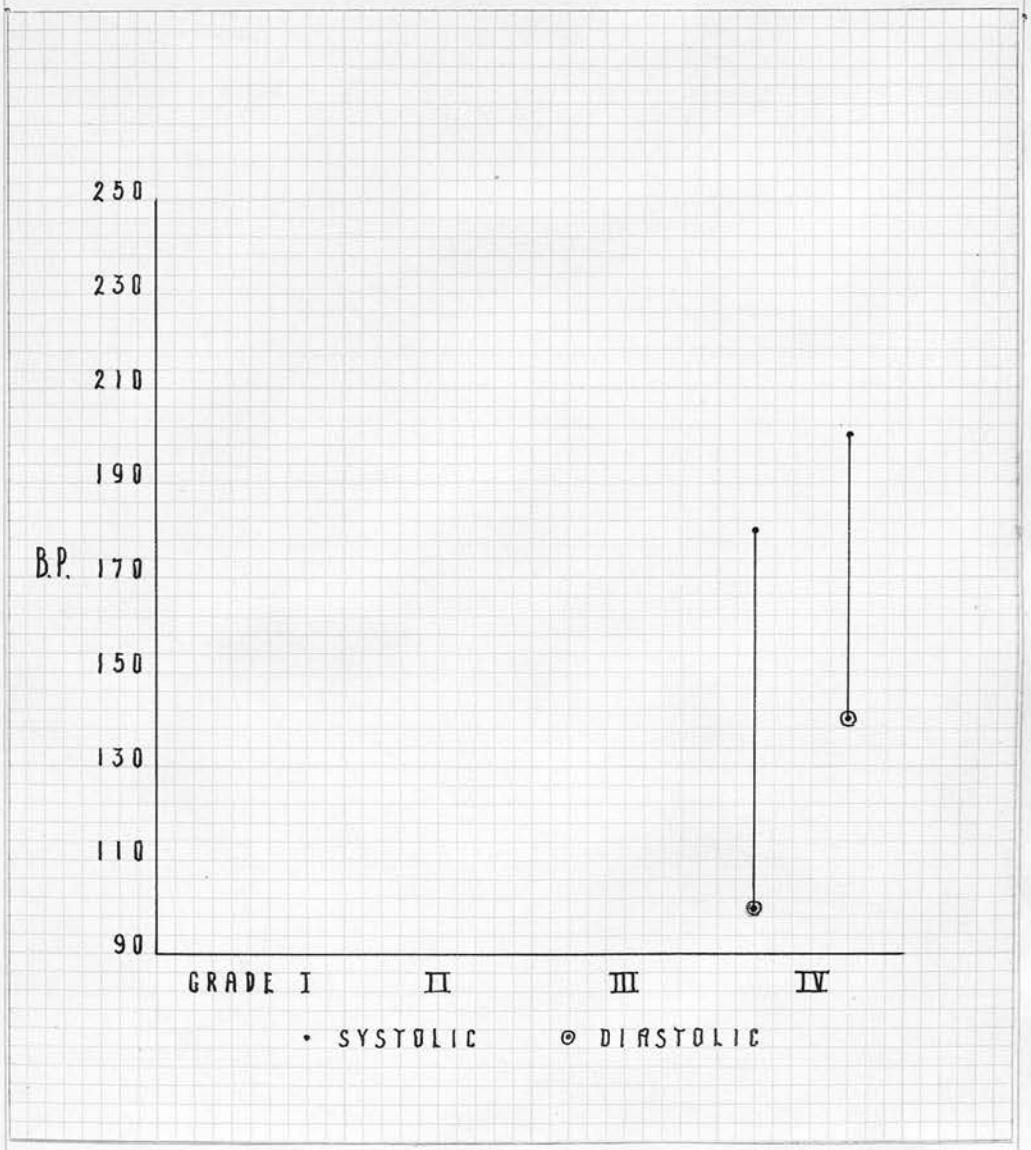


There is no correlation between the blood urea nitrogen and blood pressure levels in patient with Chronic Nephritis until the blood urea nitrogen rises above 100 mgm%., thereafter, the blood pressure rises with the blood urea nitrogen.

B. OCCIPITAL HEADACHE.

1. B.P. levels and fundal changes.

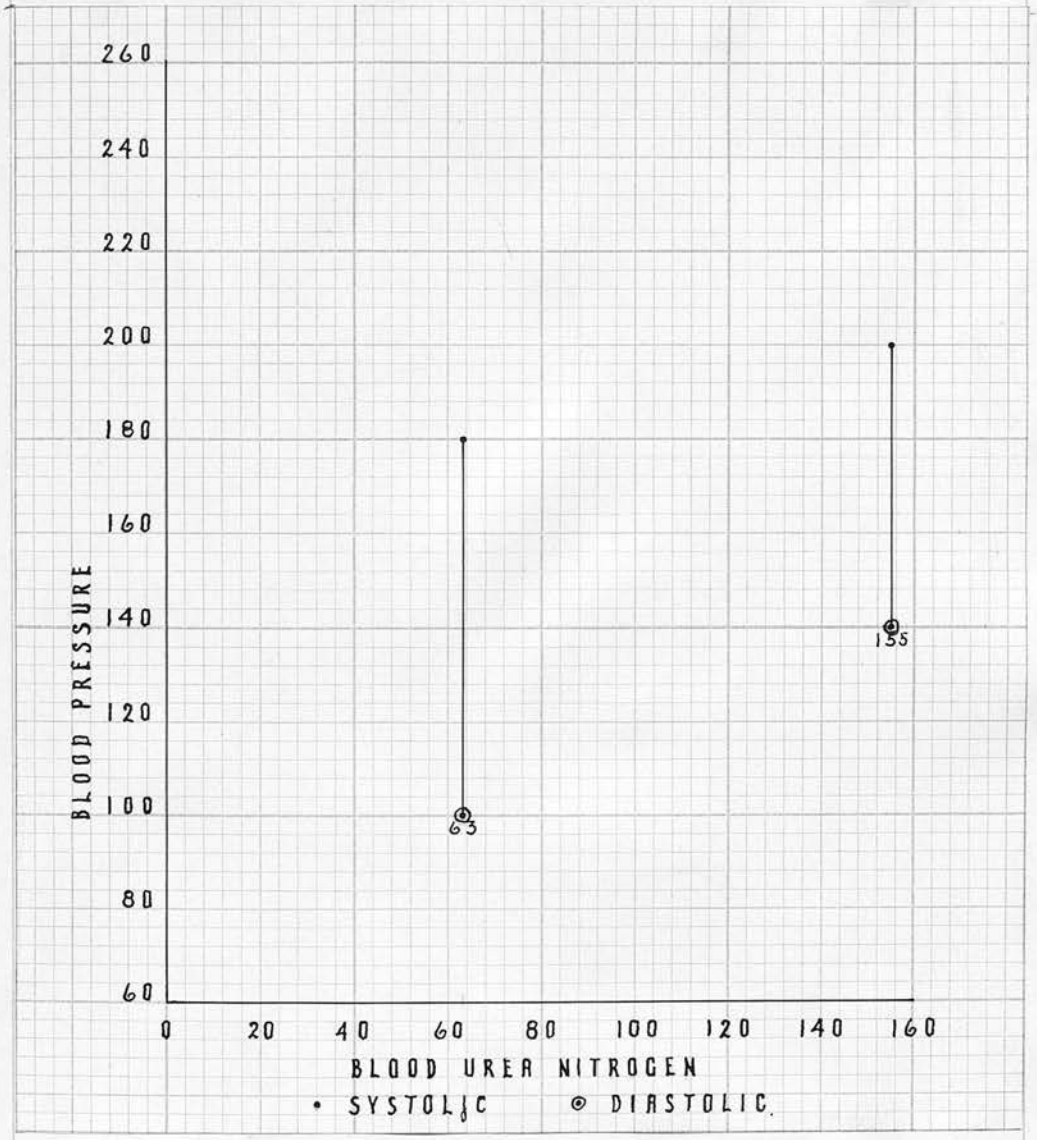
Diagram XXXI shows the levels of blood pressure with the corresponding fundal changes.



Except that the two patients with occipital headache had grade IV. fundal changes, there is no correlation.

2. B.P. levels and Blood Urea Nitrogen.

Diagram XXXII. shows the relationship between the height of the blood pressure and the level of the blood urea nitrogen.



In these 2 cases, although a few in number, there is a correlation between the B.P. levels and the blood Urea Nitrogen.

Preliminary Conclusions of the relationship between the pathological changes in patients with Frontal and Occipital Headache.

(Diagrams XXVIII. to XXXII.

A. Frontal Headache:

1. Blood pressure levels and fundal changes:

This suggests but does not show definitely a correlation between the blood pressure levels and the fundal changes.

2. Fundal Changes and Blood Urea Nitrogen :

There is a definite correlation between the blood urea nitrogen and the fundal changes. The higher the level of blood urea nitrogen, the more severe are the fundal changes.

3. Blood Urea Nitrogen and the B.P. levels:

There is no correlation between the blood urea nitrogen and the blood pressure levels, until the blood urea nitrogen rises above 100 mgm%., thereafter the blood pressure rises with the blood urea nitrogen.

B. Occipital Headache:

1. B.P. Levels and Fundal Changes:

Except that the two patients with occipital headache had grade IV. fundal changes, there is no correlation.

2. Fundal Changes and Blood Urea Nitrogen:

Too small a number (only two) to draw a graph, but both are grade IV., and the blood urea nitrogen is 63 and 155 mgm%., thereafter, it suggests a relationship between the fundal changes and the blood urea nitrogen.

3. Blood Pressure levels and the Blood Urea Nitrogen.

In these two cases, although a few in number, there is a correlation between the blood pressure levels and the blood urea nitrogen. The higher the blood urea nitrogen, the higher the blood pressure levels.

The Relationship between the pathological changes in both groups as compared with Table XXI.

	B.P. and Fundi	B.P. and Blood Urea Nitrogen.	Fundi and Blood Urea Nitrogen.
Frontal Headache	-	-	+
Occipital Headache	-	+	+

Relationship of the Severity of the Headache with Pathological Changes in Chronic Glomerulo-Nephritis.

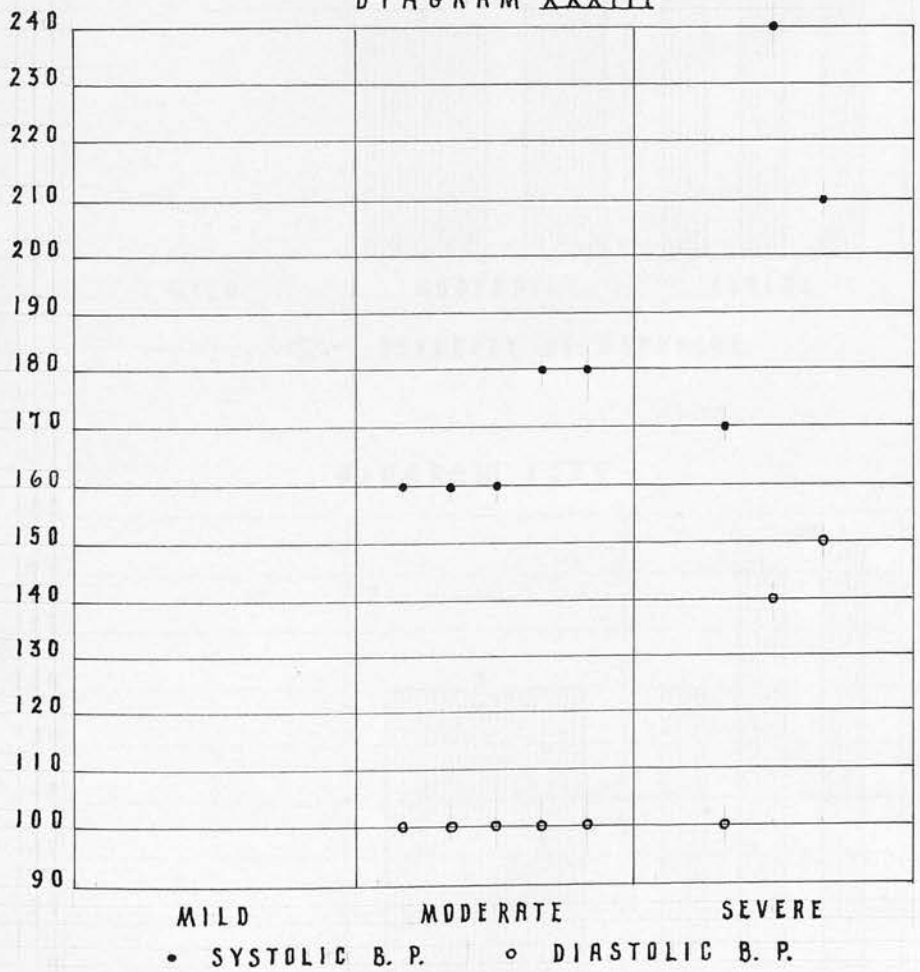
As before, the headache is divided clinically into frontal and occipital.

A. Frontal Headache:

1. Severity of frontal headache and blood pressure levels:

Diagram XXXIII. shows the relationship between the severity of the headache and the levels of blood pressure.

DIAGRAM XXXIII



The range of blood pressure can be seen in the following Table:

Severity of Headache.	The range of Systolic Pressure.	The range of Diastolic Pressure.
Moderate	160 - 180	100
Severe	170 - 240	100 - 150

When the severity of the Frontal headache is compared with the levels of the systolic and diastolic pressure separately as in diagrams XXXIV and XXXV. it can be seen that the range of both systolic and diastolic pressure was greater when the headache was severe. Thus the severity of the headache is dependent on both the systolic and diastolic pressure.

DIAGRAM XXXIV

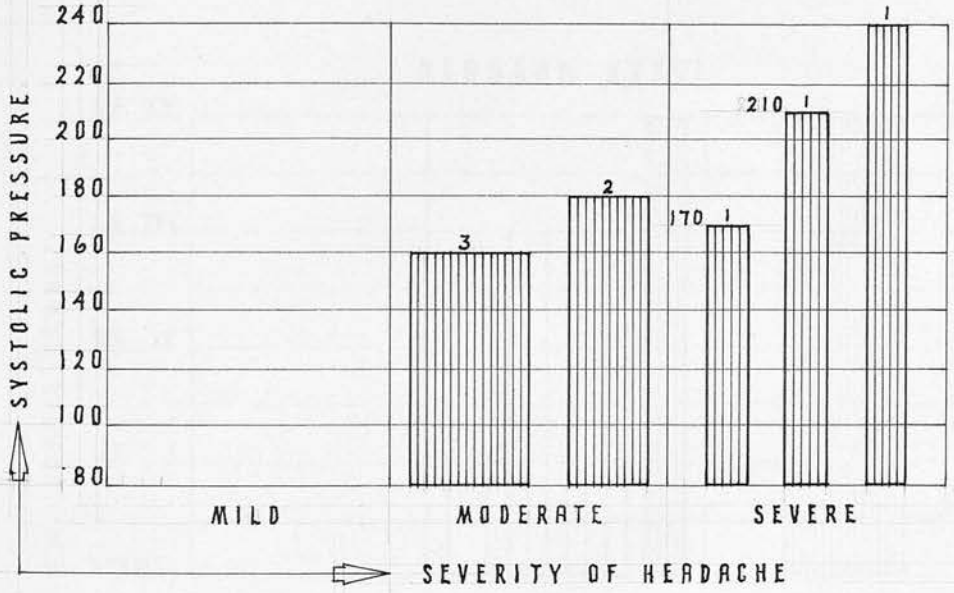
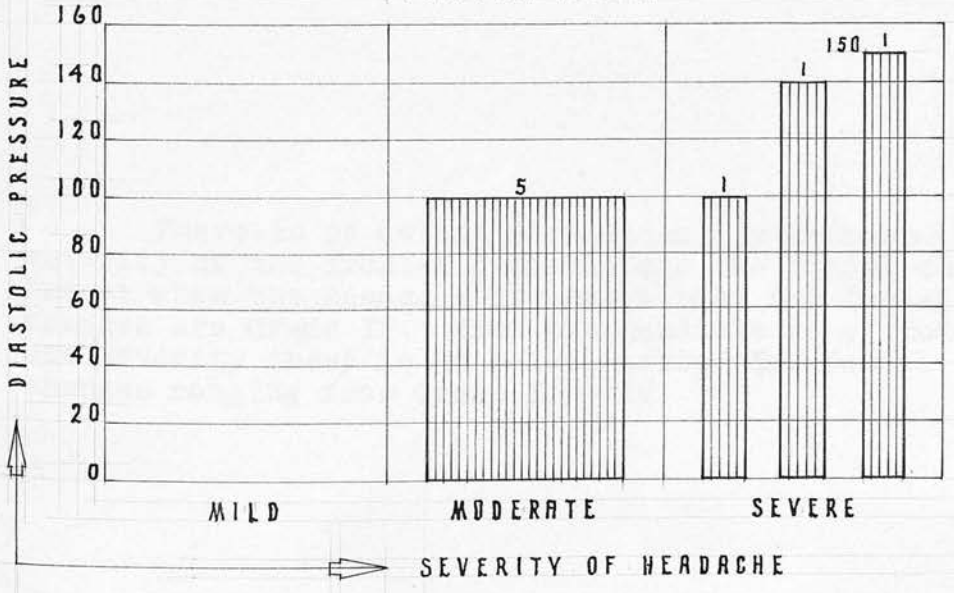


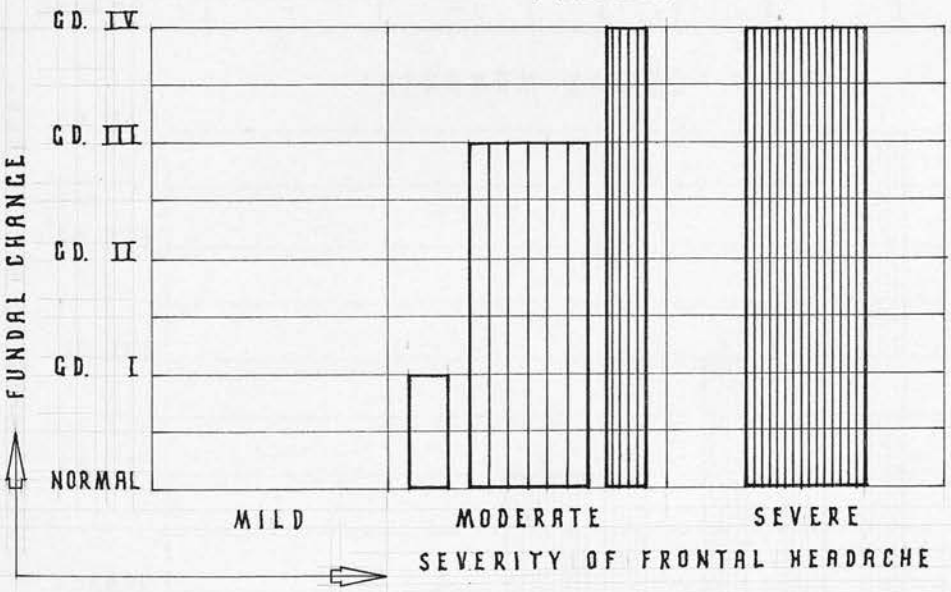
DIAGRAM XXXV



2. Severity of Frontal Headache and Fundal Changes:

The fundal changes, gradedaccording to the classification of Wagner and Keith, have been compared with the severity of the headache in Diagram XXXVI.

DIAGRAM XXXVI



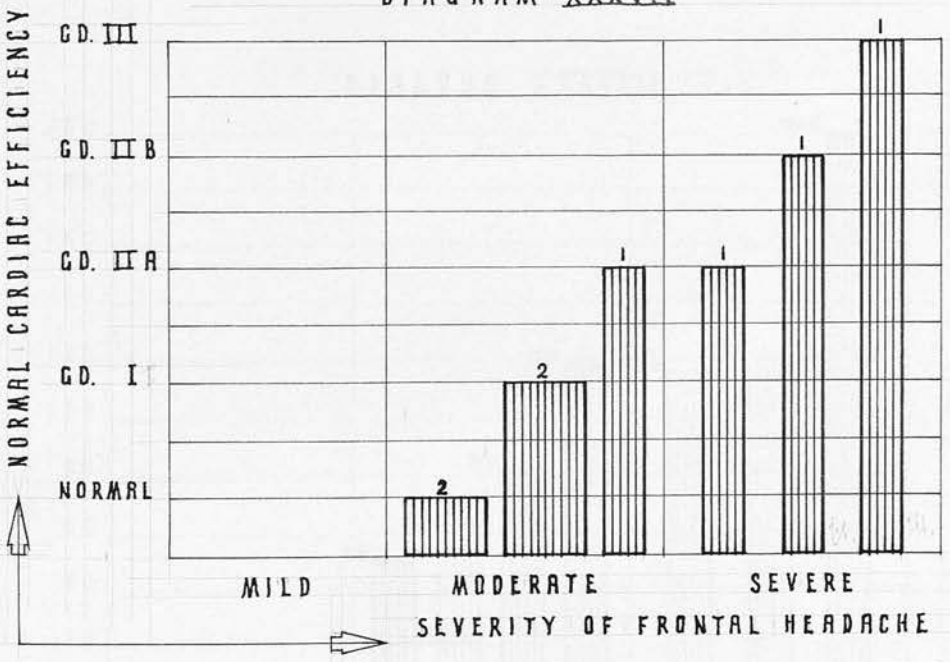
There is no definite relationship between the severity of the frontal headache and the fundal changes. Except when the headache is severe then the fundal changes are Grade IV. When the headache is of moderate severity there is no relationship, the fundal changes ranging from Grade I. - IV.

3. Severity of Frontal Headache and Cardiac Efficiency:

The severity of the frontal headache is compared with the cardiac efficiency in Table XXIII. and also in Diagram XXXVII.

Severity of Headache.	Normal	Grade I.	Grade IIA.	Grade IIB.	Grade III.
Mild	-	-	-	-	-
Moderate	2	2	1	-	-
Severe	-	-	1	1	1

DIAGRAM XXXVII



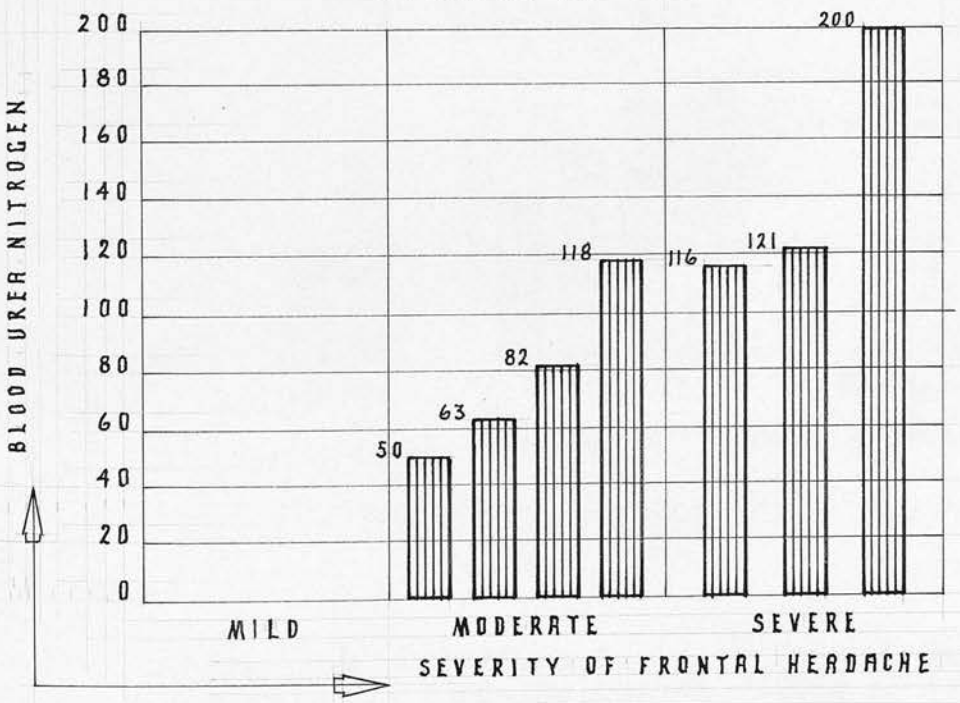
It can be seen that the severity of the frontal headache bears a direct relationship to the degree of cardiac decompensation. The more severe the headache, the greater the cardiac decompensation.

4. Severity of the Headache and Blood Urea Nitrogen:

The severity of the frontal headache is compared with the blood urea nitrogen in diagram XXXVIII.

Severity of Frontal Headache	Range of Blood Urea Nitrogen
Mild	-
Moderate	50 - 118 mgm%.
Severe	116 - 200 mgm%.

DIAGRAM XXXVIII

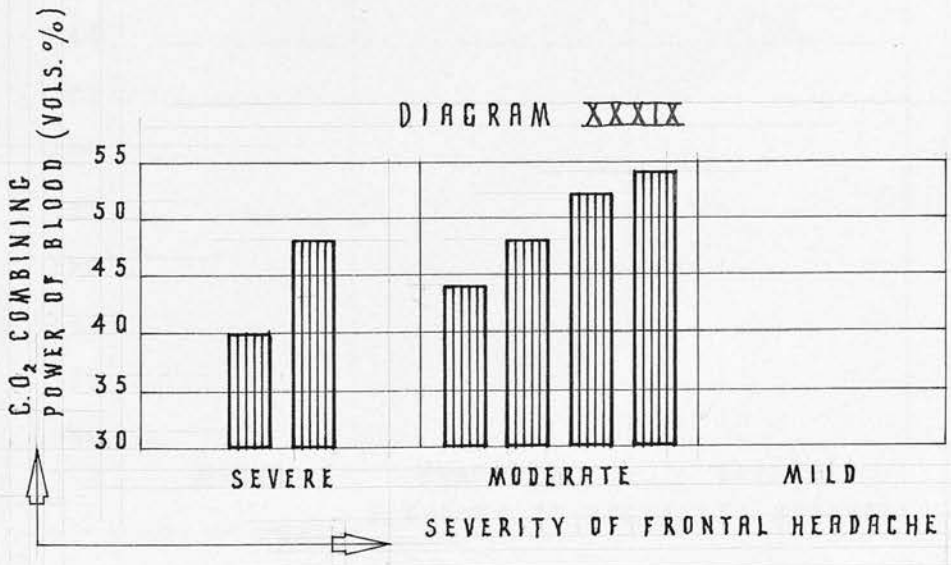


In this series, there was one patient who had a blood urea nitrogen of 200 mgm%., and experienced a severe degree of headache. Apart from that one case, there is no relationship between the severity of the headache and the level of blood urea nitrogen in the blood.

5. Severity of Frontal Headache and Co_2 Combining Power of the blood:

The severity of the headache is compared with the Co_2 combining power of the blood in Diagram XXXIX.

Mild	
Moderate	44 Vols%., 48, 44, 54.
Severe	40, 48 Vols%.



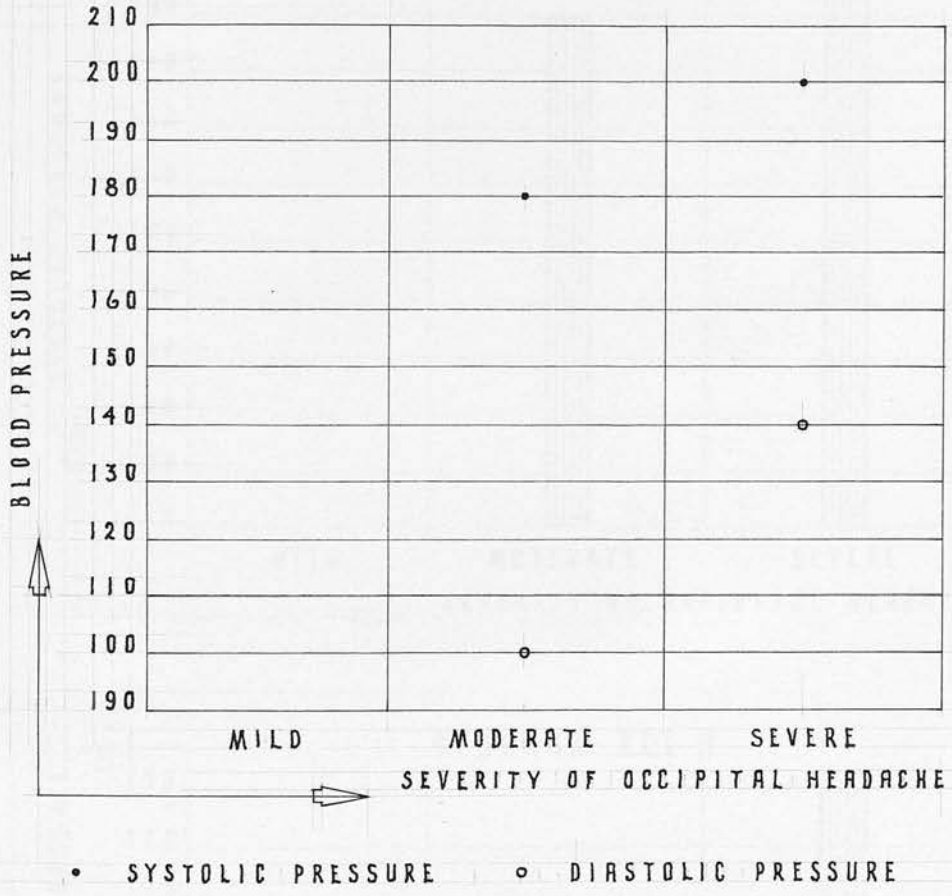
It can be seen that there is a relationship between the severity of the headache and the degree of acid-aemia as measured by the Co_2 combining power.

B. Occipital Headache:

1. The Severity of Headache and Blood Pressure Levels.

The severity of the occipital headache is compared with the blood pressure levels in Diagram XL.

DIAGRAM XL



• SYSTOLIC PRESSURE ○ DIASTOLIC PRESSURE

Severity	Range of Systolic Pressure.	Range of Diastolic Pressure.
Moderate	180	100
Severe	200	140

The relationship between the severity of the occipital headache and the systolic and diastolic pressure as seen in Diagrams XLI. and XLIA.

DIAGRAM XLI

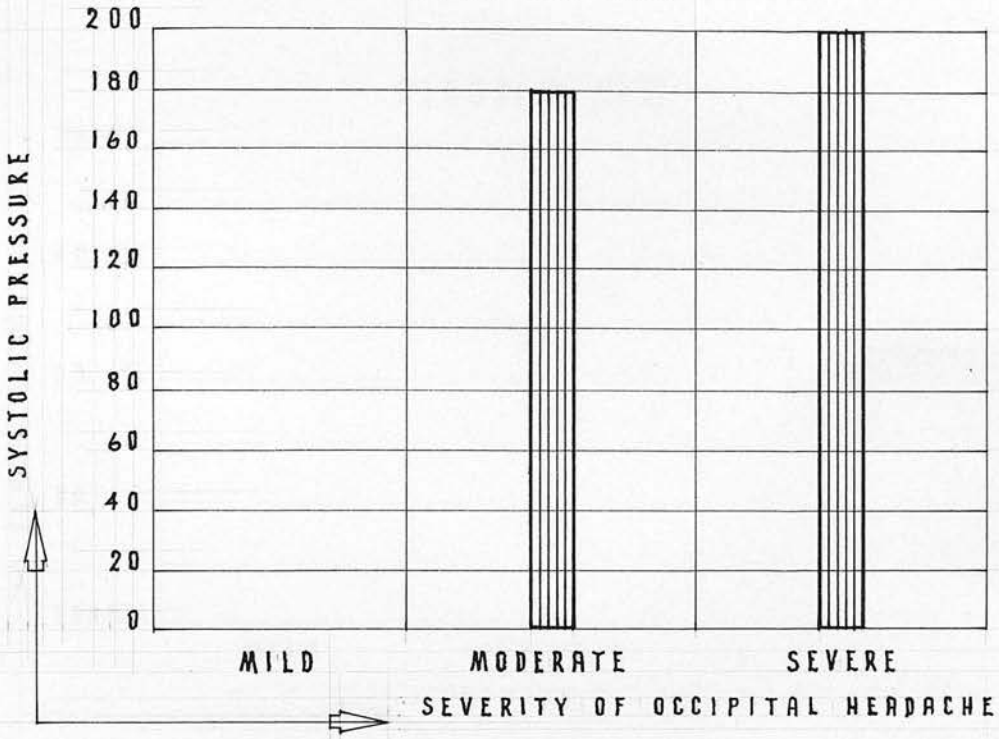
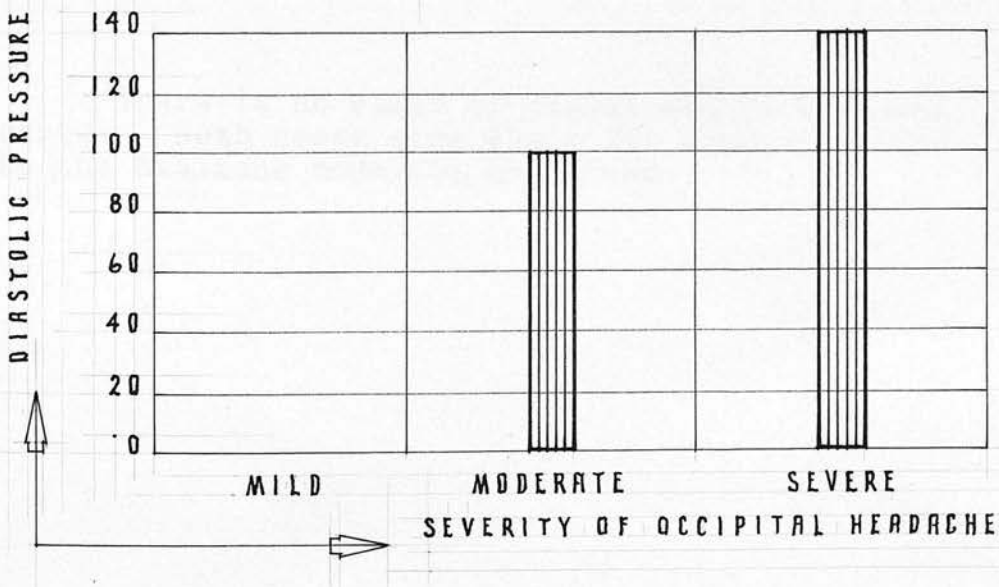


DIAGRAM XLI A

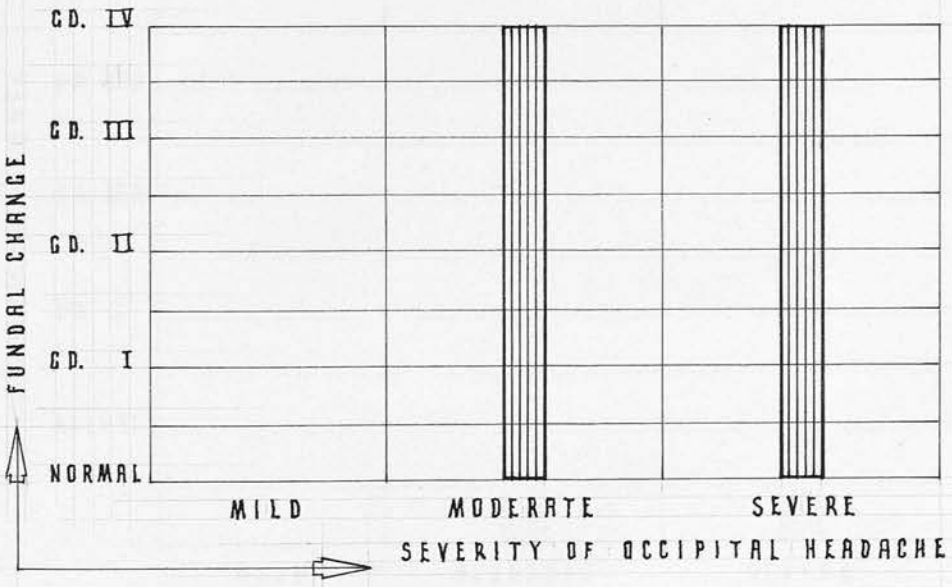


These diagrams show that the severity of the headache, is directly related to the height of the Systolic and Diastolic Pressure.

2. The severity of the Occipital Headache and Fundal Changes:

The severity of occipital headache is compared with the corresponding fundal changes in Diagrams XLII.

DIAGRAM XLII



There is no range of fundal change in these cases. Both cases show Grade IV. fundal changes, be the headache moderate or severe.

3. The severity of the headache and Cardiac Efficiency:

The severity of the occipital headache is compared with the cardiac efficiency in Table and Diagram XLIII.

DIAGRAM XLIII

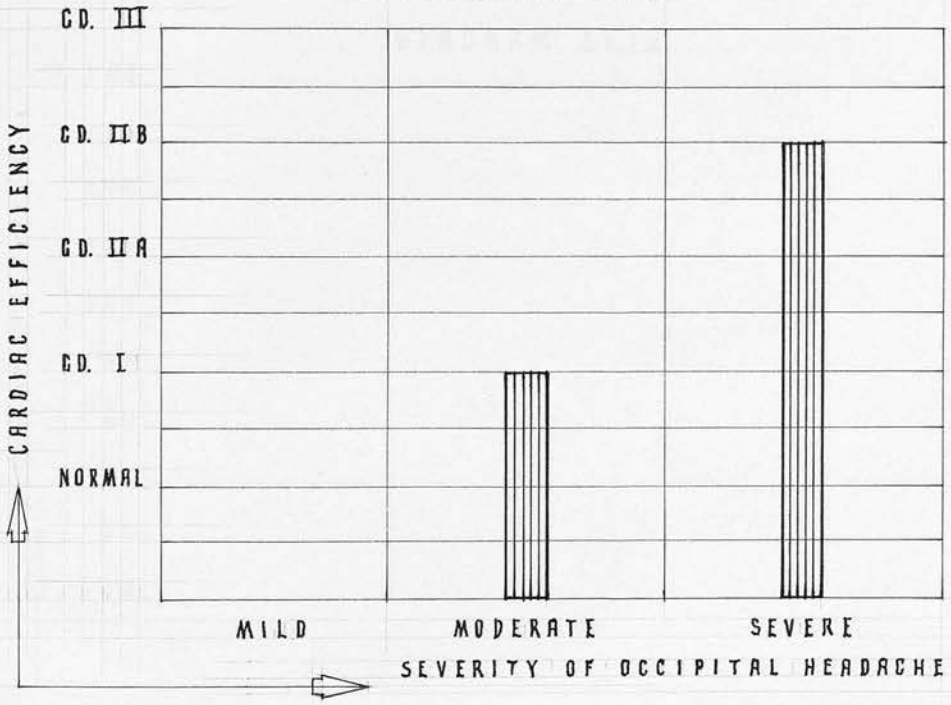


Table XXIV.

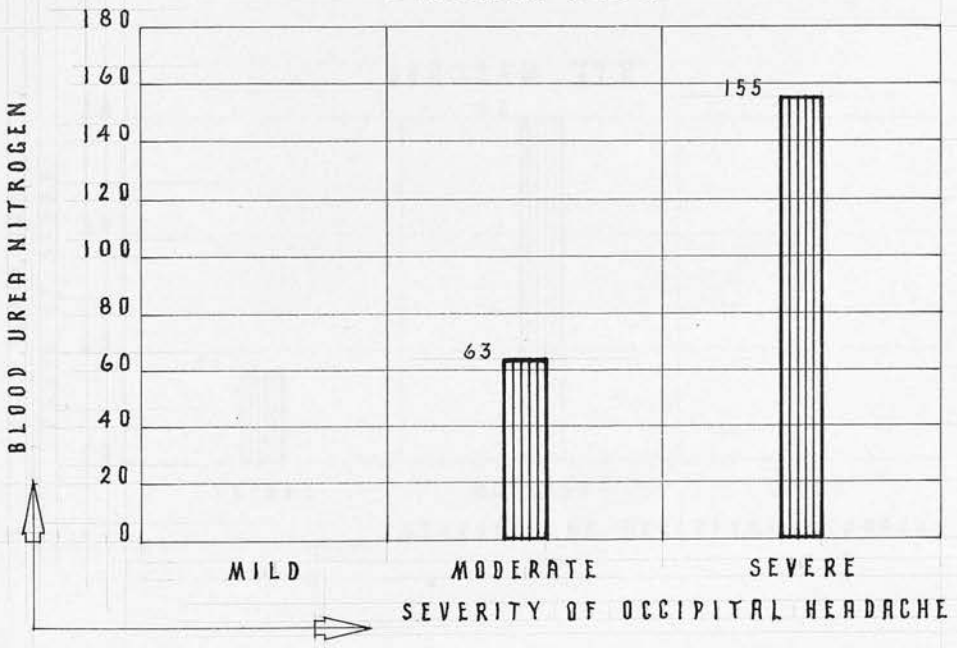
Severity of Headache	Normal	Grade I.	Grade IIA.	Grade IIB.	Grade III.
Mild	-	-	-	-	-
Moderate	-	1	-	-	-
Severe	-	-	-	1	-

The severity of the occipital headache shows a direct relationship to the degree of cardiac decompensation.

4. The Severity of Headache and Blood Urea Nitrogen:

The severity of occipital headache is compared with the corresponding levels of the blood urea nitrogen in Diagram XLIV.

DIAGRAM XLIV

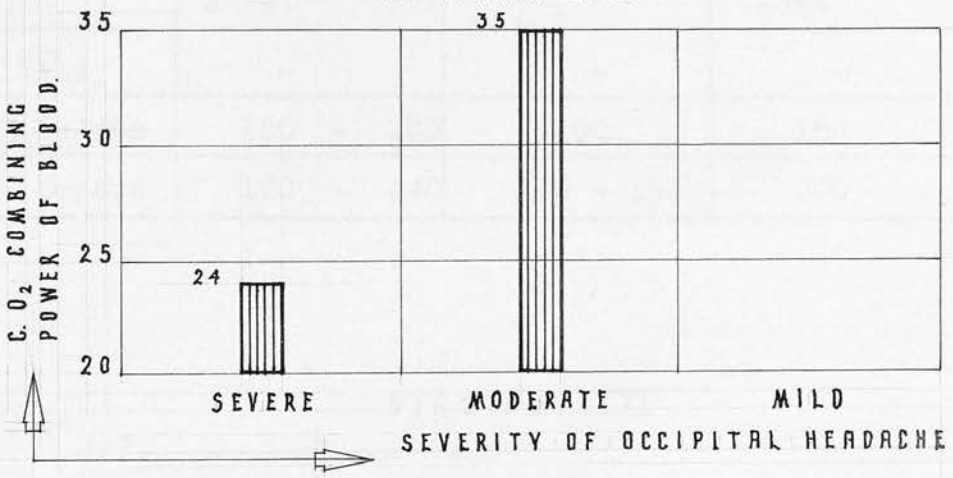


There is a definite relationship between the severity of the headache and the level of Blood Urea Nitrogen.

5. The severity of headache and Co_2 Combining Power of the Blood :

The severity of occipital headache is compared with the Co_2 combining power of the blood, in Diagram XLV.

DIAGRAM XLV



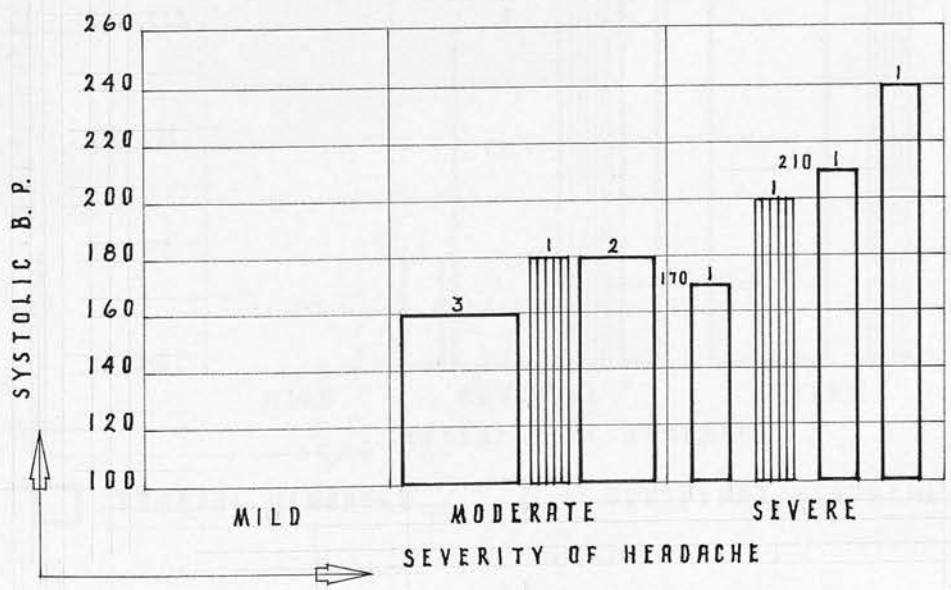
There is a direct relationship between the severity of headache and the degree of acidaemia as measured by the Co_2 combining power.

Comparison of Severity of Frontal and Occipital headache and the relationship to Blood Pressure, Fundi, Cardiac Efficiency, Blood Urea Nitrogen and Co_2 Combining power of the blood.

1. Comparison of the severity of frontal and occipital headache, with the level of blood pressure in Table XXV. and Diagrams XLVI. and XLVII.

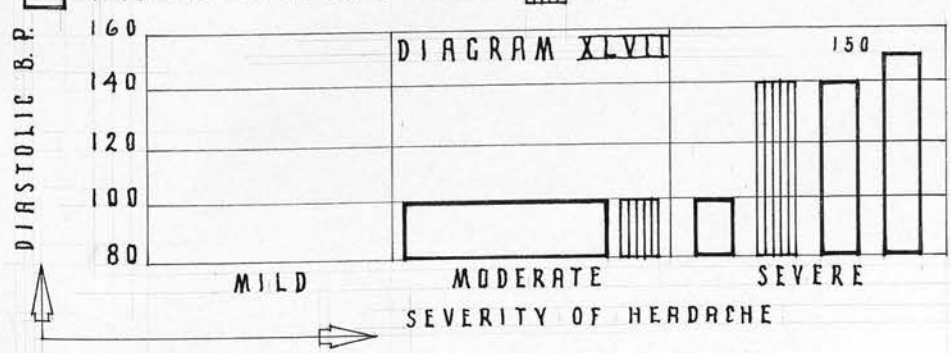
	Frontal		Occipital	
Severity of Headache.	Range of Systolic Pressure.	Range of Diastolic Pressure	Range of Systolic Pressure	Range of Diastolic Pressure.
Mild	-	-	-	-
Moderate	160 - 180	100	180	100
Severe	170 - 240	100 - 150	200	140

DIAGRAM XLVI



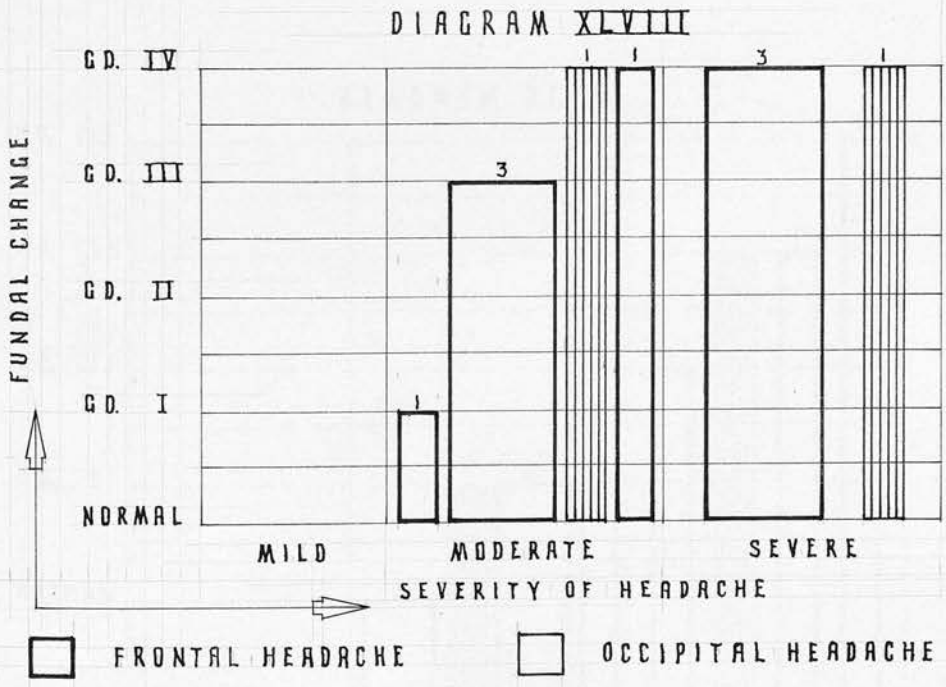
FRONTAL HEADACHE OCCIPITAL HEADACHE

DIAGRAM XLVII



2. Comparison of the severity of fundal and occipital headache with the fundal changes in Diagram XLVIII.

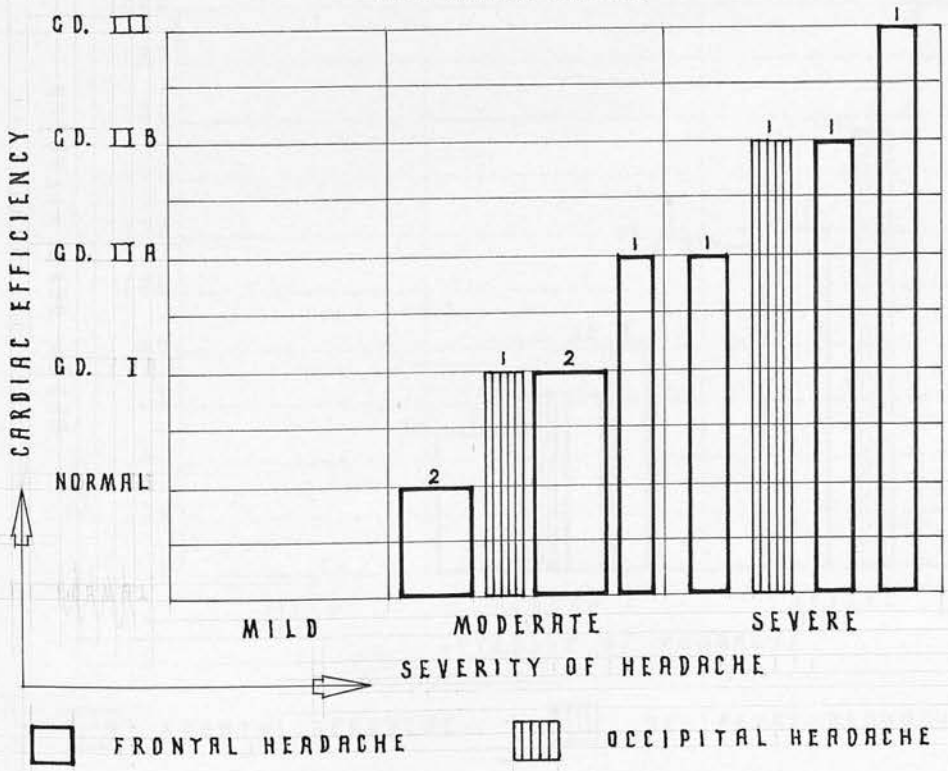
Severity of Headache	Frontal	Occipital
Mild	-	-
Moderate	Stage I. - IV.	IV.
Severe	IV.	IV.



3. Comparison of Cardiac Efficiency with the severity of Frontal and Occipital Headache in Diagram XLIX.

	Frontal					Occipital				
Severity of Headache	Normal	Grade I.	Grade IIA.	Grade IIB.	Grade III.	Normal	Grade I.	Grade IIA.	Grade IIB.	Grade III.
Mild	-	-	-	-	-	-	-	-	-	-
Moderate	2	2	1	-	-	-	1	-	-	-
Severe	-	-	1	1	1	-	-	-	1	-

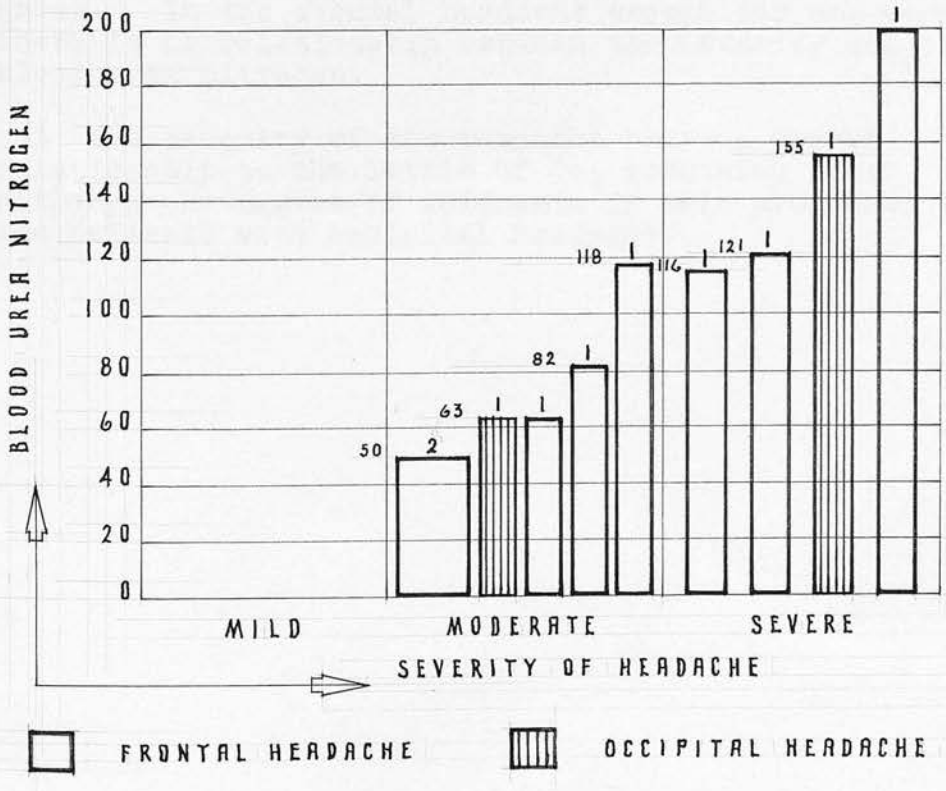
DIAGRAM XLIX



4. Comparison of Frontal Headache with Blood Urea Nitrogen and Occipital Headache and Blood Urea Nitrogen in relation to its severity in Diagram L.

Severity	Frontal	Occipital
Mild	-	-
Moderate	50-118 mgm%.	63 mgm%.
Severe	116-200 mgm%.	155 mgm%.

DIAGRAM I



Conclusions of Relationship of Pathological Changes with the Severity of Frontal and Occipital Headache.

(1) The severity of the frontal headache bears a direct relationship to the levels of systolic and diastolic pressure. The severity of the occipital headache also bears a direct relationship to these figures, although in frontal headache the blood pressure levels are much higher.

(2) The severity of the frontal and occipital headache is difficult to assess in relationship to the fundal changes. In occipital headache, headache of both moderate and severe degree show Grade IV. changes. In frontal headache, the moderate headache showed Grades I. - IV., and the severe headache Grade IV.

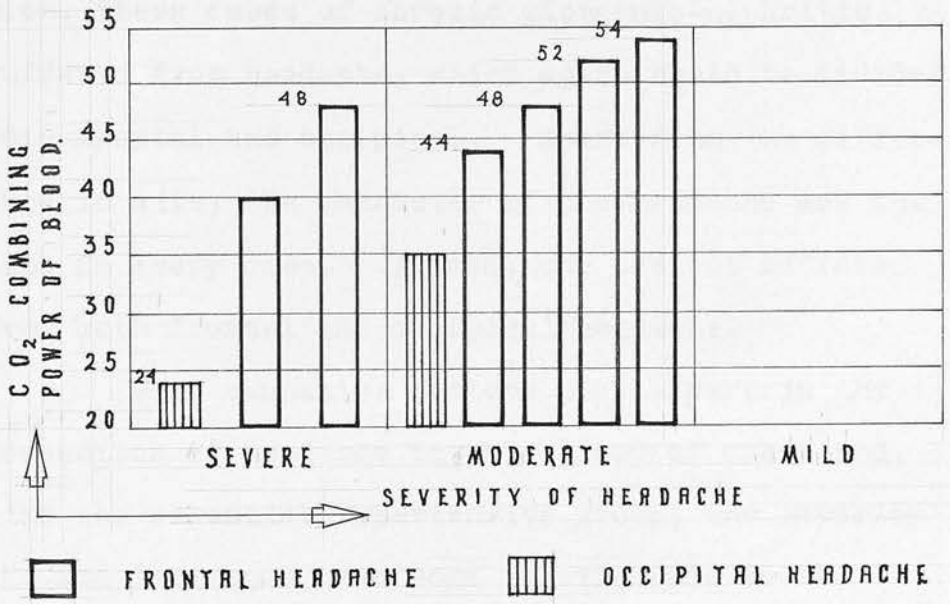
(3) The severity of the headache in both frontal and occipital headache bears a direct relationship to the cardiac efficiency.

(4) The severity of the headache and the blood urea nitrogen shows a definite relationship in the occipital group. In the frontal headache except for one case, there is no relationship between the severity and the blood urea nitrogen.

(5) The severity of the headache bears a direct relationship to the levels of Co_2 combining power. Although the degree of acidaemia is more pronounced in the patients with occipital headache.

5. Comparison of Frontal and Occipital Headache in relation to its severity with the Co_2 Combining Power of the Blood in Diagram LI.

DIAGRAM LI



Discussion of the incidence of headache in patients suffering from hypertension due to chronic glomerulo-nephritis.

As in the patients with essential hypertension, these cases of chronic glomerulo-nephritis suffered from headache, which again could be divided into frontal and occipital. Apart from the difference in site, the character of the headache was the same in every case. Indeed, one patient suffered from both frontal and occipital headache.

Many causative factors play a part in the production of headache in this group of cases and, like the essential hypertensive group, the severity and the incidence bear some relationship to the pathological changes resulting from the chronic glomerulo-nephritis.

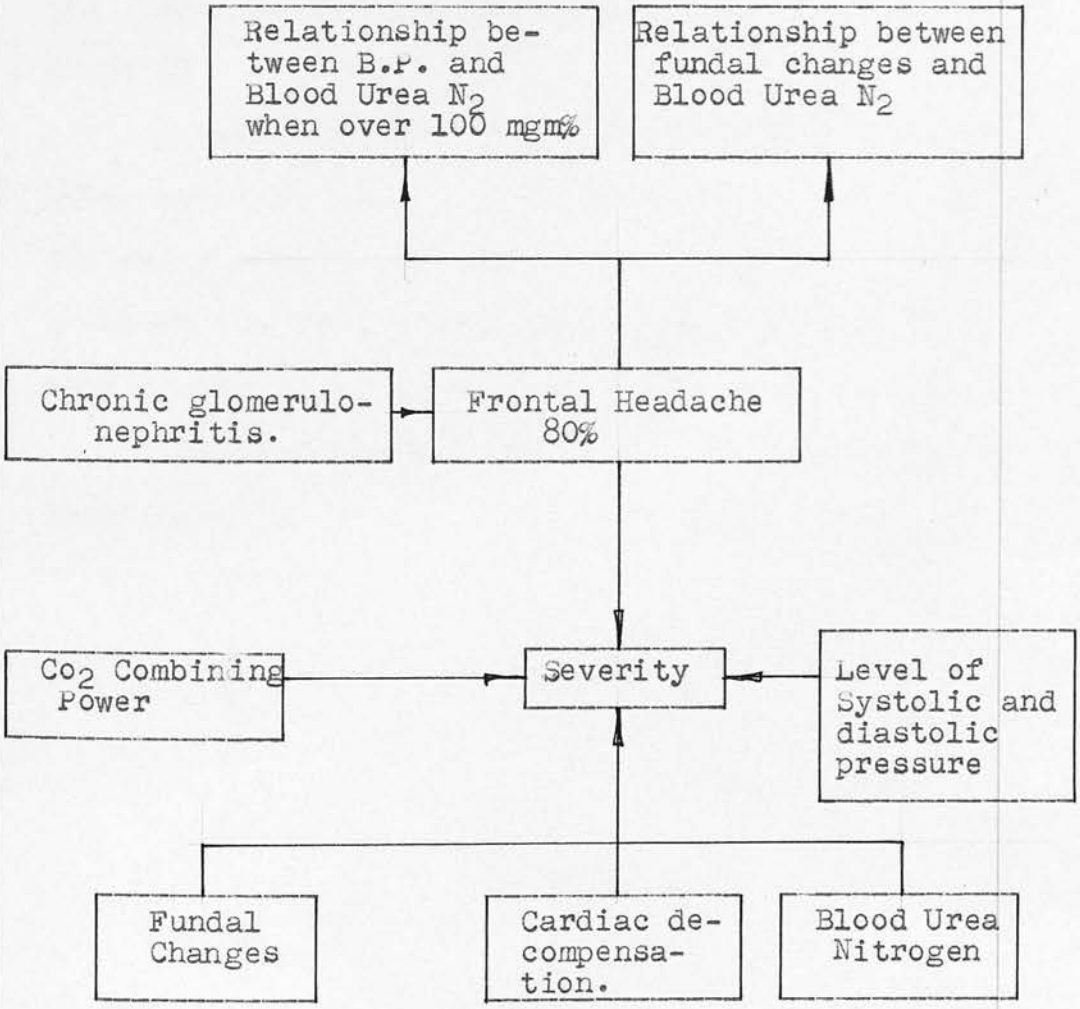
In this group, patients with frontal headache showed a relationship between the fundal changes and the level of the blood urea nitrogen, and between the blood pressure levels and the blood urea nitrogen when this latter rose above 100 mgm %. Below this figure, there was no relationship. There was no relationship between the fundal changes and the blood pressure in this group.

The severity of the frontal headache depended upon many factors, upon the blood urea nitrogen, upon the degree of acidaemia, upon the fundal changes and upon the degree of cardiac decompensation and the height of the systolic and diastolic pressure.

The severity of the headache could be classified as moderate and severe, and of the eight cases in this group, five were of moderate severity and three were severe.

These relationship are seen, perhaps, more clearly in the following diagram :-

Diagram A:-



In a similar way in the occipital headache group, there was a relationship between the blood

the blood urea nitrogen and the blood pressure levels and between the blood urea nitrogen and the fundi. There was no relationship between the levels of the blood pressure and the fundi.

The severity of the headache had a definite relationship to the level of blood urea nitrogen, to the degree of acidaemia, to the blood pressure levels and to the degree of cardiac decompensation. The fundal changes in these two cases were both Grade IV.

Although there were only two cases of occipital headache, one being of moderate severity and one being severe, the degree of acidaemia and the level of the blood urea nitrogen was much greater than in the eight patients with frontal headache.

These relationships can be represented diagramatically :-

Diagram: B:-

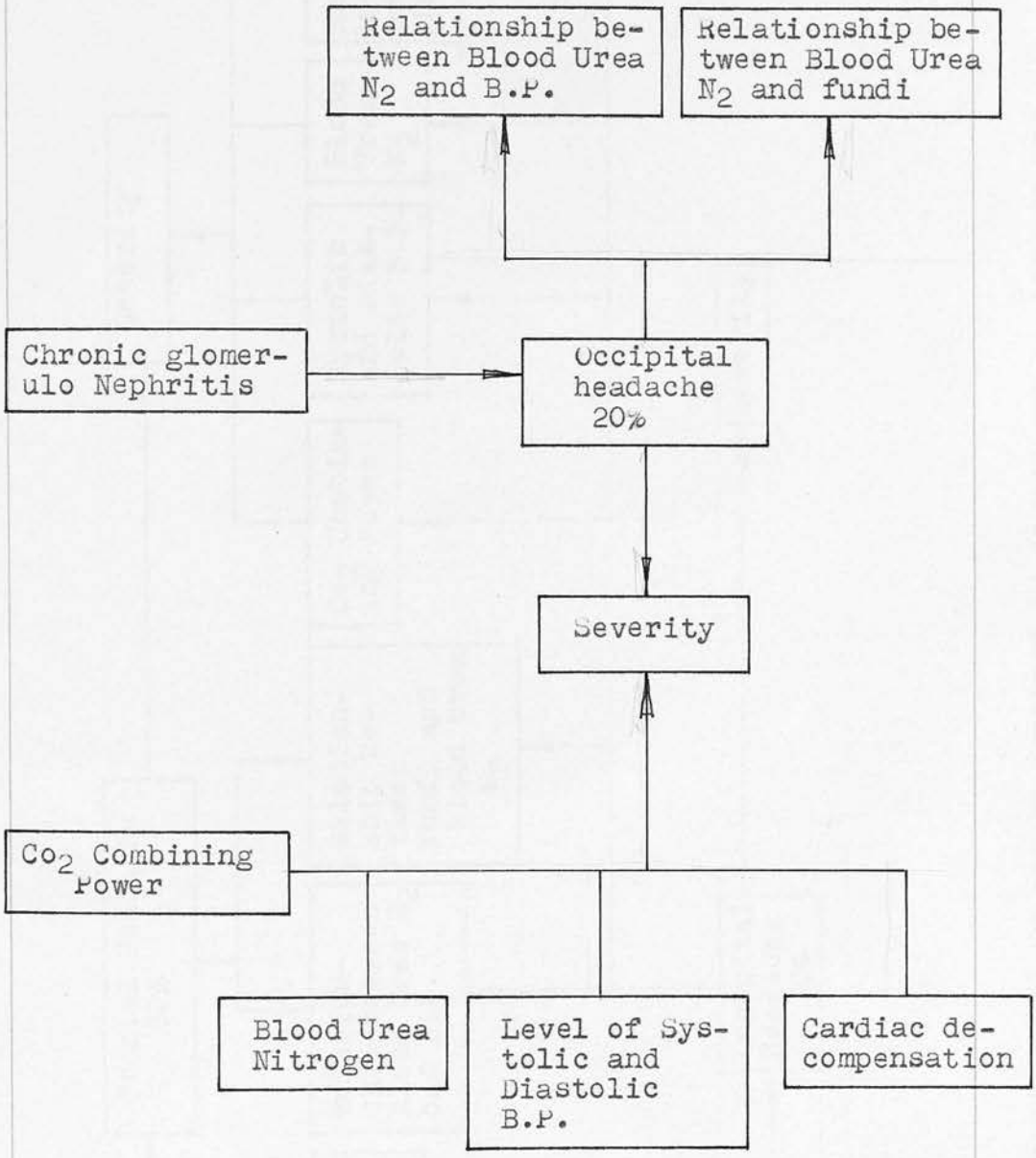
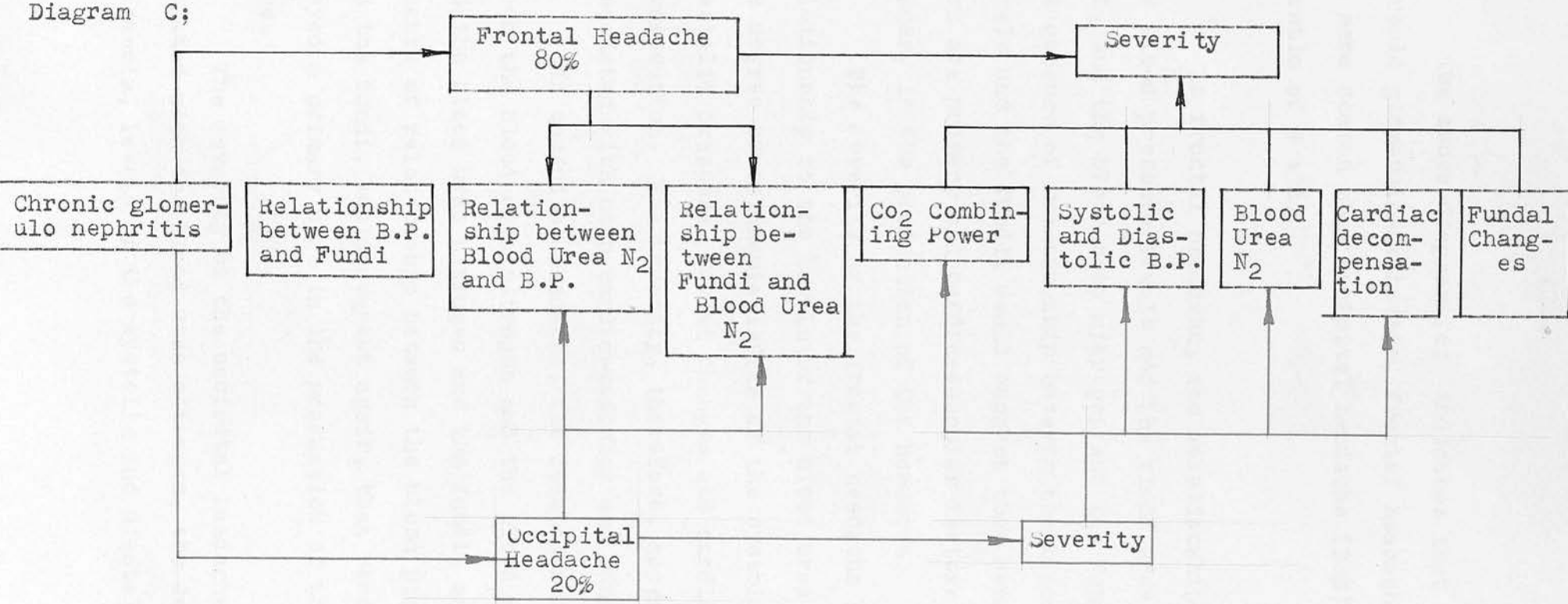

Relationship between Blood Urea N_2 and B.P.
Relationship between Blood Urea N_2 and fundi
Chronic glomerulo Nephritis
Occipital headache 20%
Severity
Co_2 Combining Power
Blood Urea Nitrogen
Level of Systolic and Diastolic B.P.
Cardiac decompensation

Diagram C;



These diagrams (A and B) can be combined in an attempt to compare and contrast the two types of headache in chronic glomerulo-nephritis - Diagram C.

The above diagram (c) indicates that in chronic glomerulo-nephritis, frontal headache (80%) is more common than occipital headache (20%). A ratio of 4 : 1.

In frontal headache, the relationship between the blood pressure levels and the blood urea nitrogen, and the blood urea nitrogen and the fundi; and the absence of relationship between the blood pressure levels and the fundi, would suggest that renal factors are primary and cardio-vascular factors are secondary in the production of the headache.

The severity of the frontal headache bears a relationship to the levels of the blood urea nitrogen, the degree of acidaemia levels of the systolic and diastolic pressure, fundal changes and cardiac decompensation. The severity, therefore, appears to be associated with both cardio-vascular and renal factors.

In occipital headache, the relationship between the blood urea nitrogen and the blood pressure, and the blood urea nitrogen and the fundi, and the absence of relationship between the blood pressure and the fundi, would suggest again, that renal factors played a primary part in the production of the headache.

The severity of the occipital headache is associated with the blood urea nitrogen, the degree of acidaemia, levels of the systolic and diastolic

pressure and the cardiac decompensation. This suggests from the height of the blood urea nitrogen and the degree of acidaemia, that renal factors mainly affect the severity of the headache, although Cardiovascular factors also play a part.

Following these observations, an attempt has to be made to suggest a cause for this type of headache seen in cases of chronic glomerulo-nephritis. When these cases are surveyed certain precipitating factors are found in each case as in the cases of essential hypertension.

Precipitating Factors and Methods of Relief :

In both groups, the majority experienced headache on rising in the morning 70%. In 20% the headache came on at any time and in 20% who experienced a constant headache, the severity was worse on rising.

The duration of the attack varied from half an hour to several hours in the patients with frontal headache and from two hours to several hours in the patients with occipital headache. The duration of the attack would be longer in the group with occipital headache. In frontal headache, the symptoms had been present from week to five years, while in occipital headache, their duration was only three to four weeks.

Relief was obtained by a variety of factors.

In the case of the frontal headache:

(1) Rest and diet gave relief in one case.
(2) Aspirin and Rest " slight relief in one case.
(3) Rest, Sodium Amytal and Aspirin gave no relief in one case.
(4) Multiple medications and Rest gave no relief in two cases.
(5) Relief obtained without medication in three cases.

In the case of the occipital headache:

(1) Rest and Aspirin gave slight relief in one case.
(2) Rest, Sodium Amytal and Aspirin gave no relief in one case.

These factors must be considered in more detail:

A. Dealing first with the frontal headache:

If one deals first with the frontal headache, then like the essential hypertensive group, in 70% of these cases, the occurrence of the headache on rising from bed in the morning, would suggest (1) sleep and (2) sudden movement again played some part.

(1) Sleep:

The same changes in the body which occur in sleep and which were discussed under the hypertensive group, are present in these patients. The changes are modified by the effects of the chronic renal insufficiency.

(2) Sudden movement:

The changes in the body produced by sudden movement will be identical with the changes discussed in the essential hypertension group, except that here again, they may be modified by the presence of the chronic glomerulo-nephritis.

In chronic glomerulo-nephritis, the changes in the body are a combination of :

(1) Renal failure.
(2) Secondary hypertension and its effects.

As mentioned earlier, the effects of renal failure are:

(a) Acidosis.
(b) Alteration of osmotic pressure.
(c) Salt depletion.
(d) Retention of nitrogenous substances.

The effects of hypertension can be tabulated thus as before :-

(1) Hypertensive heart.
(2) Increased coronary blood flow.
(3) Arteriosclerosis.
(4) Reduced cardiac output.
(5) Increased venous pressure.
(6) Reduced in oxygen tension.
(7) Increased Co_2 tension.

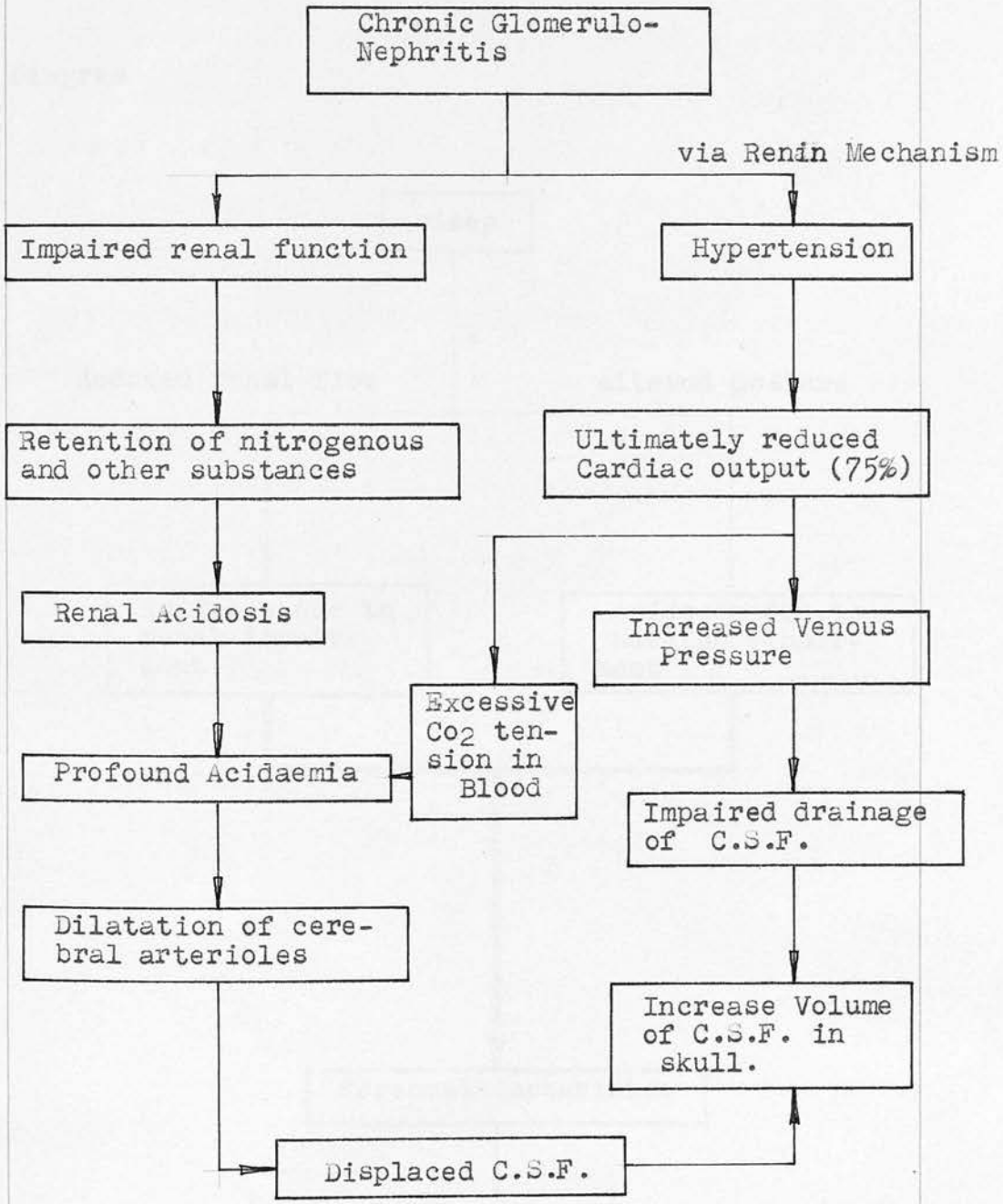
Sleep as mentioned earlier, (page 91) produces acidaemia and a lowering of oxygen in the blood, an increased Co_2 tension and a fall in the blood pressure.

A combination of renal impairment with the cardiac decompensation (seen in 6 out of 8 cases - 75%) results in further acidaemia. The ultimate effect of cardiac decompensation, producing increased venous pressure, causes impaired drainage of cerebro-spinal fluid and this, together with the effects of acidosis and retention of nitrogenous substances results in marked dilatation of the cerebral arterioles together with an increased volume of cerebro-spinal fluid in the skull.

These changes can be represented diagramatically:

The Effects of Renal Impairment.

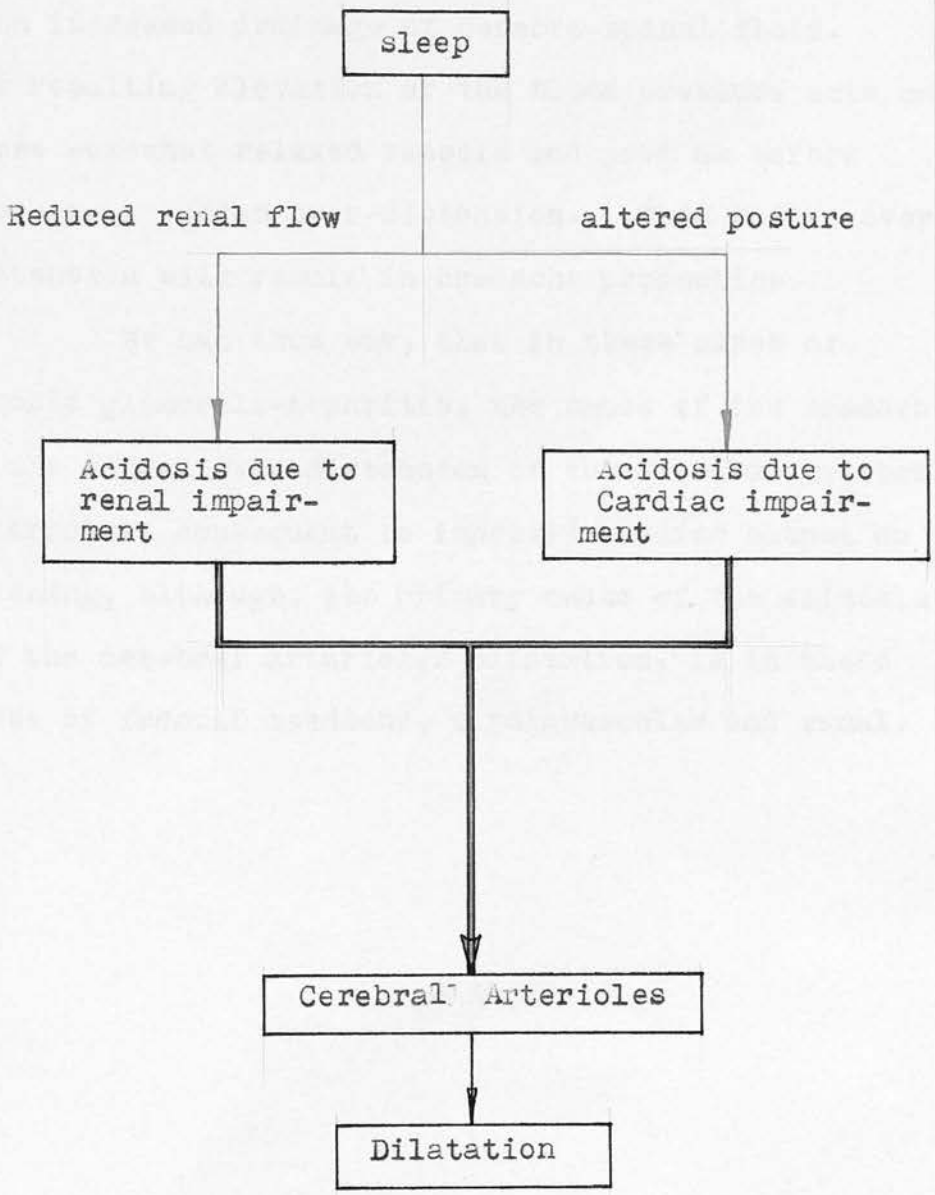
Diagram:



The effect of sleep in these cases is to dilate further the cerebral vessels; for the acidosis is increased due to reduced renal blood flow and further impairment of cardiac efficiency.

This can be represented diagramatically :

Diagram



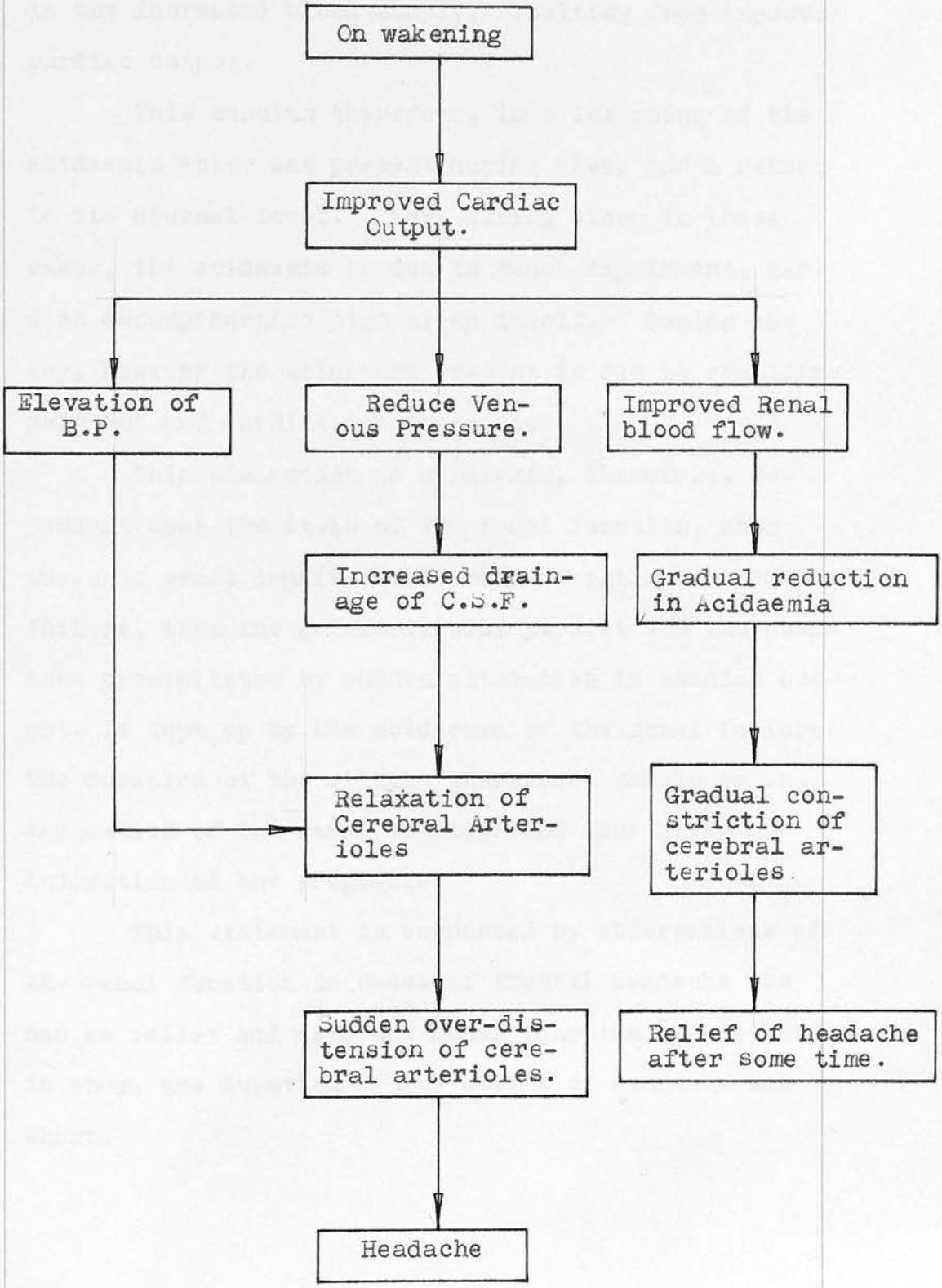
Mechanism on headache production on wakening:

On wakening, just as in the group with essential hypertension, there is an increased cardiac output with resulting improvement of blood flow through the kidneys and a quickening of respiration - both these factors reduce the acidaemia. The improved cardiac output produces a reduction in venous pressure with increased drainage of cerebro-spinal fluid. The resulting elevation of the blood pressure acts on these somewhat relaxed vessels and just as before produces a sudden over-distension. This sudden over-distension will result in headache production.

We can thus say, that in these cases of chronic glomerulo-nephritis, the cause of the headache is the sudden over-distension of the dilated cerebral arterioles, consequent to improved cardiac output on wakening, although, the primary cause of the acidosis and the cerebral arteriolar dilatation, is in these cases of frontal headache, cardiovascular and renal.

The effects on wakening:

Diagram:



The relief of the headache may be produced by vaso-constriction, resulting from diminution in acidaemia. For this, further impairment of renal function due to sleep, is improved on wakening due to the increased blood supply, resulting from improved cardiac output.

This results therefore, in a lessening of the acidaemia which was present during sleep and a return to its diurnal level. For, during sleep in these cases, the acidaemia is due to renal impairment, cardiac decompensation plus sleep itself. During the day, however the acidaemia present is due to renal impairment and cardiac decompensation.

This diminution in acidaemia, therefore, dependent upon the state of the renal function, when there is gross impairment of renal function, or renal failure, then the acidaemia will persist and the headache precipitated by sudden alteration in cardiac output, is kept up by the acidaemia of the renal failure. The duration of the attack, therefore, should be an indication of the renal function and thus gives an indication of the prognosis.

This statement is supported by observations of the renal function in cases of frontal headache who had no relief and with the renal function of patients in whom, the duration of the attack of headache was short.

In this series, three patients received no therapy to relieve their headache. In these cases, the duration of the headache, when compared with renal function gives a surprising correlation.

Table XXVI.

		Blood Urea Nitrogen		
	Duration of attack	on admission	on discharge	Urea - Range
Case I.	More than 2 hours.	118 mgm%	21 mgm%	sp.gr. 1018 - 2 gms% of urea " " 1012 - 1·2 " "
Case II.	About 1 hour	50 mgm%	30 mgm%	" " 1016 - 2·4 " " " " 1010 - 1·2 " "
Case III.	About 1 hour	50 mgm%	30 mgm%	" " 1016 - 2·5 " " " " 1010 - 1·2 " "

In all the above cases, the renal function improved in hospital, indicating renal impairment only and not renal failure.

It can be seen however, that there is a direct relationship between the duration of the attack of headache and the renal function. The more severe the renal impairment, the longer the attack of headache. These three patients suffered from renal impairment as indicated by the level of the blood urea nitrogen on admission and at this time they suffered from headache of moderate severity but of average duration (1½ hours). With improvement in renal function, the duration of the headache lessened until at the time of discharge, the attacks of headache had abated.

It is interesting to compare the above resulte (Table XXVI) with the patients in this series, who died from renal failure and who obtained no relief from headache in spite of therapy.

Table XXVII.

	Duration of Attack	Blood Urea Nitrogen	Urea-Range	Post-Mortem findings
Case I.	Several hours	116 mgm% did not fall.	died before carried out	Chronic glomerulo-Nephritis.
Case II.	Several hours.	121 mgm% did not fall.	sp.gr. 1010 - 1 gm% 1010 - 0·5	Chronic glomerulo-Nephritis.
Case III.	Several hours.	200 mgm% did not fall.	died before carried out.	Chronic Glomerulo-Nephritis.

Persistence of the headache, therefore, would suggest a bad prognosis, indicating renal failure and persistence of the acidaemia, in spite of improved cardiac output on wakening.

Relief of Frontal Headache in Chronic Glomerulo-Nephritis.

In these cases patients experienced relief by:-

(1) Rest and suitable diet for the nephritic condition gave relief in 50%.

(2) Aspirin alone gave only slight relief in 12%.

(3) Sodium Amytal combined with Aspirin gave no relief in 12%.

(4) No relief was obtained in spite of multiple medication in 25%.

1. Rest and suitable diet:-

The effect of rest in these patients will be similar to that in the series suffering from essential hypertension. Rest will limit movement, there will be no sudden alterations in cardiac output and thus no sudden over-distension of the dilated cerebral arterioles and thus the mechanism of headache production will not occur.

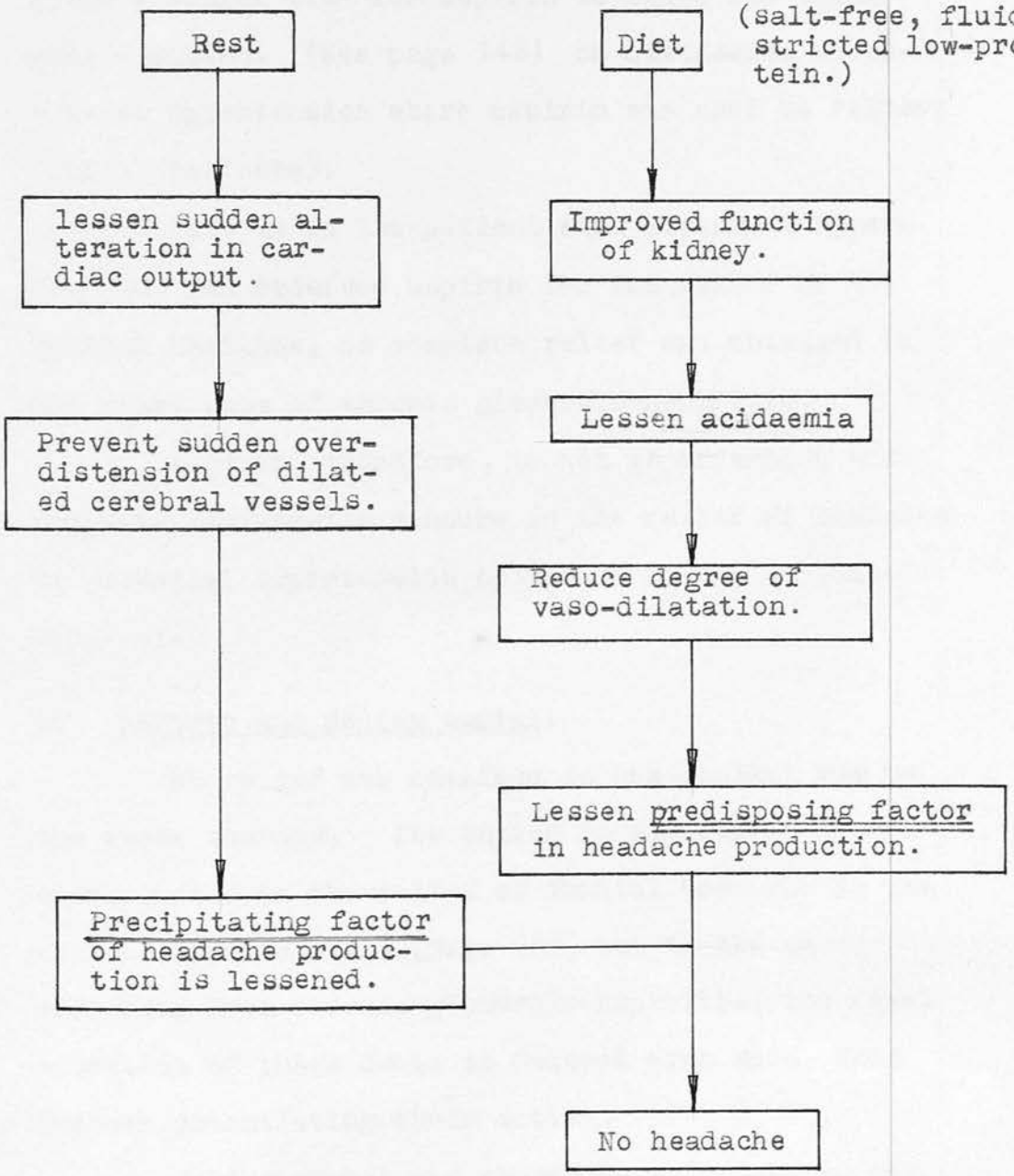
Furthermore by dietetic measures, such as salt restriction, restricted fluid intake and a protein intake of initially only 50 grammes,the work of the kidney would be reduced and its function rapidly restored to normal. The renal academia,therefore, one factor in the production of cerebral arteriolar dilatation, is lessened.

Both, rest and dietetic treatment would therefore prevent sudden changes in cardiac output,with over-distension of the cerebral vessels; the acidaemia would be lessened with reduction in the degree of cerebral arterioles dilatation.

See diagram :-

The effect of Rest and Diet in relief of frontal headache in Chronic Glomerulo-Nephritis:

Diagram



2. Aspirin:

Aspirin alone gave slight relief in one patient. In renal impairment, the excretion of salicylate in the urine is delayed. This, therefore, gives a longer time for aspirin to exert its therapeutic action. (See page 148) on discussion on essential hypertension where aspirin was used to relieve frontal headache).

Just as in the patient with essential hypertension, who received aspirin for the relief of his frontal headache, no complete relief was obtained in the above case of chronic glomerulo-nephritis.

Aspirin, therefore, is not an effective and complete therapeutic measure in the relief of headache in essential hypertension nor in chronic glomerulo-nephritis.

3. Aspirin and Sodium Amytal:

No relief was obtained in one patient due to the above therapy. Its action is similar to the cases quoted in the relief of frontal headache in essential hypertension, (page 118) but in the case, suffering from chronic glomerulo-nephritis, the renal excretion of these drugs is delayed even more, thus further potentiating their action.

Sodium amytal was chosen in this case as the headache was severe and sodium amytal's action is much more rapid than is luminal.

In spite of the actions of both aspirin and sodium amytal, no relief from the frontal headache was obtained in this case.

This patient died from Uraemia. This fact bears out the previous observation, that headache of long duration unrelieved by any therapy has a bad prognosis.

Two other patients with severe frontal headache experienced no relief in spite of many drugs being tried. Both these patients died. These three patients are the cases quoted previously who had severe frontal headache for several hours. (See Table XXVII.& cases Nos. 18, 23, 24.

In this series of cases, frontal headache, therefore, which fails to respond to any therapy, always indicates a bad prognosis - the patients dying of Uraemia.

Preliminary conclusions based on patients with frontal headache in Chronic Glomerulo-Nephritis.

(1) Frontal headache is a common symptom in chronic glomerulo-nephritis and occurred in 80% in this series.

(2) It is always throbbing in character.

(3) It usually occurs on rising in the morning from bed and if present throughout the day, is made worse by sudden movement.

(4) Relief was obtained in very few cases by the administration of drugs.

(5) From the effects of renal failure and secondary hypertension and the modification of these changes produced by sleep; the mechanism of headache production would appear to be, the sudden over-distension of dilated cerebral arterioles. The sudden over-distension is produced by alterations in cardiac output and the blood pressure levels, while the initial vasodilatation is the result of the acidaemia of impaired renal function plus cardiac decompensation and the physiological acidaemia of sleep.

(6) The severity of the frontal headache is associated with the renal function, the cardiac decompensation, fundal changes, the levels of the systolic and diastolic blood pressure and the degree of acidaemia. This acidaemia results from the increase in Co_2 tension of the heart failure plus the retained substance due to renal impairment.

(7) The duration of the attack of frontal headache gives an indication as to the renal function and ultimate prognosis,. The longer the attack, the more impaired the renal function and the worse the prognosis.

(9) The failure to respond to any therapy also gives an indication of renal function and prognosis. Severe renal failure and death from uraemia occurred in patients who experienced no relief from the various types of therapy prescribed.

Occipital Headache in Chronic Glomerulo-Nephritis.

In this series, two patients complained of occipital headache (20%). One patient, however, had both occipital and frontal headache. Although there were only two patients with occipital headache, the severity of the attack and the gross changes found on examination inthese cases are significant and warrant further attention.

Our previous findings show a relationship between the severity of the occipital headache and the levels of systolic and diastolic blood pressure, cardiac decompensation, renal impairment and the acidaemia. In these cases, there is a relationship between the blood urea nitrogen and the blood pressure levels and between the blood urea nitrogen and the fundal changes. There is no correlation between the blood pressure and the fundal changes.

As before, the patient with occipital headache, only experienced the attack on rising.

The patient who experienced occipital headache only will be considered first.

In this case (case No. 22), no relief was obtained and the patient died from Uraemia. He had a blood urea nitrogen of 155 mgm% and a Co_2 combining power of 24 Vols%. His fundi showed grade IV. changes and his urine had a sp. gr. of 1010 and contained blood and granular casts. The blood pressure was $\frac{200}{140}$, and there were minor signs of cardiac decompensation. (Enlarged heart, mitral systolic murmur, accentuated second aortic sound and E.C.G.; left Adis deviation).

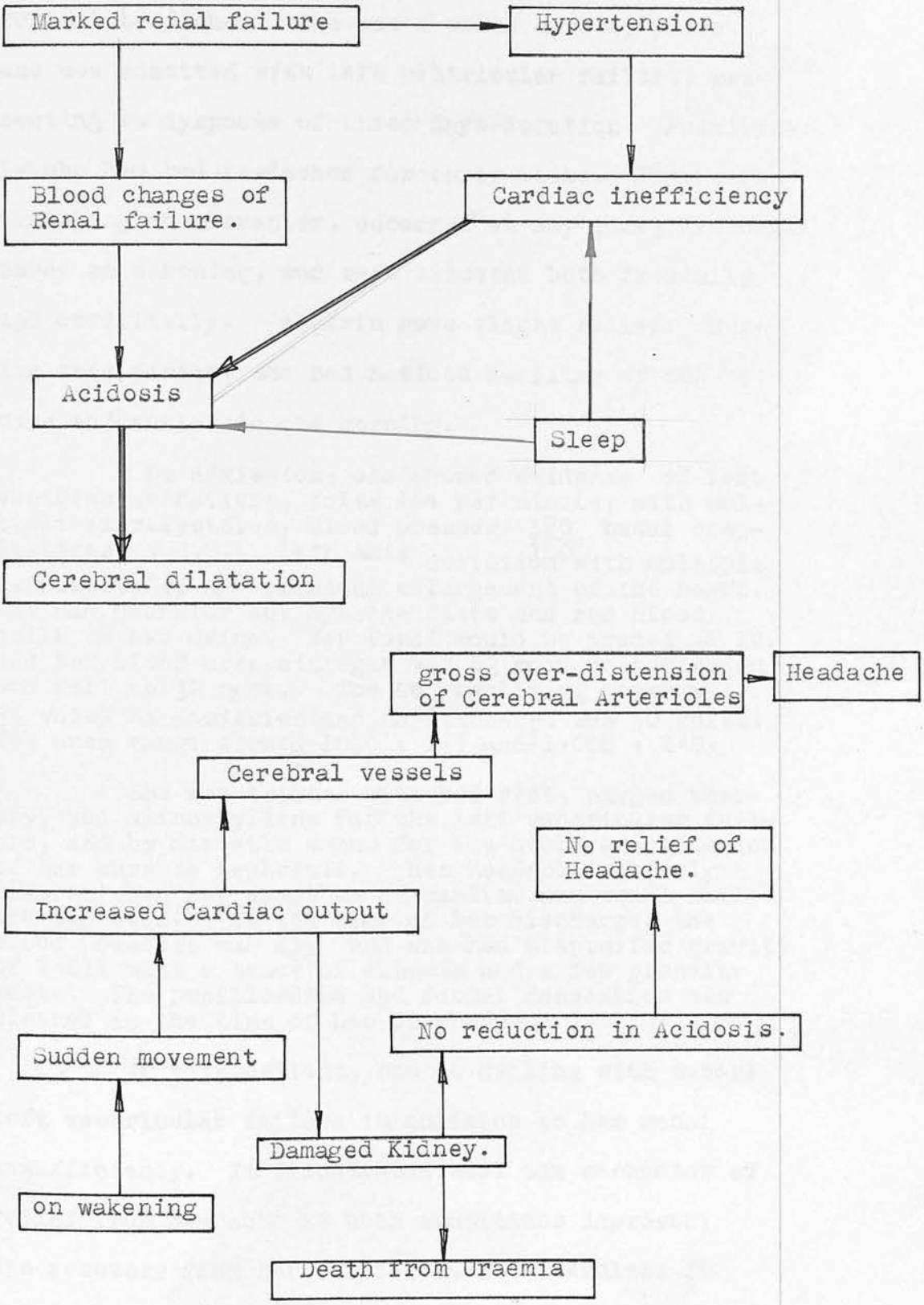
The severe acidosis from this man's renal failure (as revealed by the lowered Co_2 combining

power) would produce vaso-dilatation. This would be made worse during sleep. The sudden alteration in cardiac dynamics produced on rising would cause over-distension of the already dilated cerebral arterioles and result in headache.

The improved blood supply to the kidneys, which should reduce the acidosis on wakening and relieve the headache, would not take place here, for the kidneys were grossly damaged and the patient was in renal failure. Furthermore, no relief was obtained by any therapy, again indicating the severity of the headache and a bad prognosis.

These changes can be represented diagramatically:

Diagram:


Marked renal failure
Hypertension
Blood changes of Renal failure.
Cardiac inefficiency
Acidosis
Sleep
Cerebral dilatation
gross over-distension of Cerebral Arterioles
Headache
Cerebral vessels
No relief of Headache.
Increased Cardiac output
No reduction in Acidosis.
Sudden movement
Damaged Kidney.
on wakening
Death from Uraemia

The second patient suffered from both occipital and frontal headache. She was a woman aged 45 years. She was admitted with left ventricular failure, presenting as dyspnoea of three days duration. Previously she had had headaches for three weeks. They were throbbing in character, occurred at any time, although worse on wakening, and were situated both frontally and occipitally. Aspirin gave slight relief. During this period, she had noticed swelling of the face and ankles in the morning.

On admission, she showed evidence of left ventricular failure, pulse 164 per minute, with multiple extrasystoles, Blood pressure $\frac{180}{100}$, basal crepitations, E.C.G.; left Axis deviation with multiple extrasystoles and clinical enlargement of the heart. She had granular and hyaline casts and red blood cells in her urine. Her fundi could be graded as IV. and her blood urea nitrogen was 63 mgm% on admission and fell to 32 mgm%. The Co_2 combining power was 35 Vols% on admission and on discharge was 50 Vols%. The urea range showed 1·010 : 1·5 and 1·008 : 1·2.

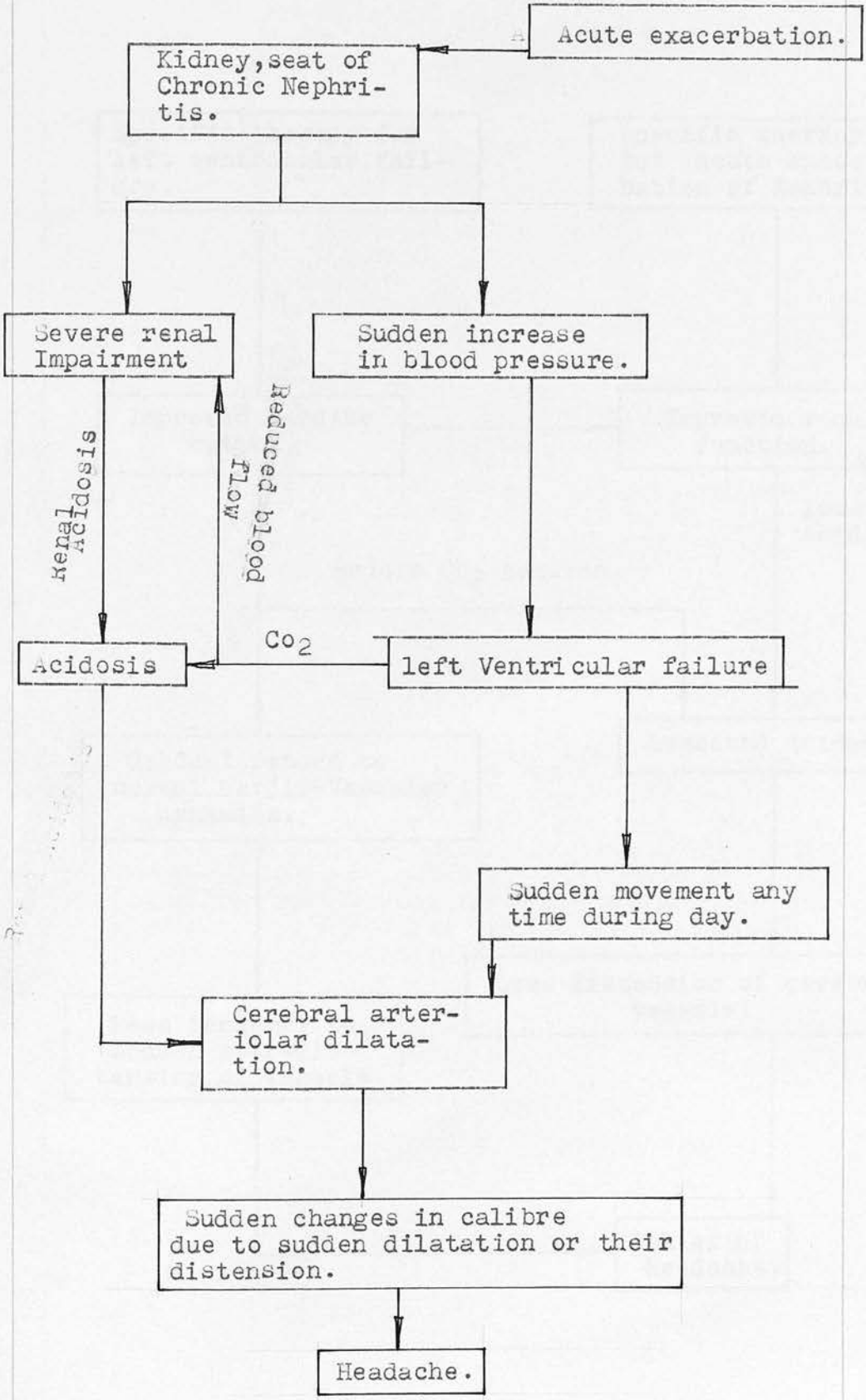
She was treated with bed rest, oxygen therapy, and aminophylline for the left ventricular failure, and by dietetic means for the acute exacerbation of her chronic nephritis. Her headache entirely cleared when her symptoms of cardiac and renal failure improved. At the time of her discharge, the blood pressure was $\frac{150}{90}$ and she had a specific gravity of 1·011 with a trace of albumin and a few granular casts. The papilloedema and fundal congestion had cleared at the time of her discharge.

In this patient, one is dealing with severe left ventricular failure in addition to her renal insufficiency. It illustrates well the mechanism of relief from headache as both conditions improved. The recovery from her cardiac failure resulted in lowering of the Co_2 tension of the blood and thus a lessening of the acidaemia. The recovery from her

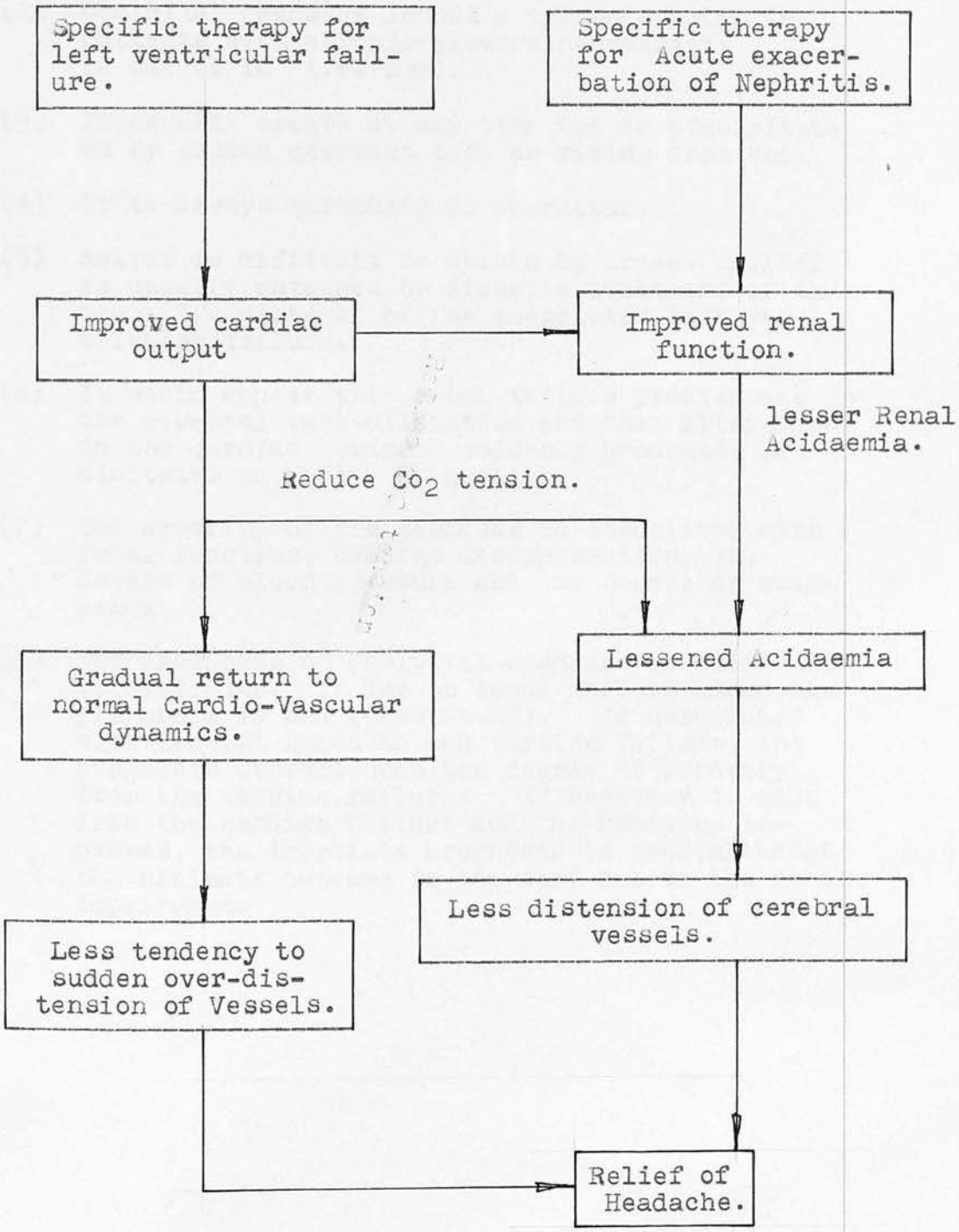
heart failure was gradual and therefore there were no sudden fluctuation in cardiac output, producing over-distension of cerebral vessels and headache. The acidaemia of the renal impairment with resulting vaso-dilatation lessened as the kidney condition was treated and thus the cerebral vaso-dilatation became less. Both cardiac and renal factors, therefore, played a part in the production of the headache and its disappearance as their respective functions improved.

As before, the mechanism and relief of headache can be represented diagramatically.

Diagram :


Acute exacerbation.
Kidney, seat of Chronic Nephritis.
Severe renal Impairment
Sudden increase in blood pressure.
Renal Acidosis
Reduced blood Flow
Acidosis
Co_2
left Ventricular failure
Sudden movement any time during day.
Cerebral arteriolar dilatation.
Sudden changes in calibre due to sudden dilatation or their distension.
Headache.

Diagram


Specific therapy for left ventricular failure.
Specific therapy for Acute exacerbation of Nephritis.
Improved cardiac output
Improved renal function.
lesser Renal Acidaemia.
Reduce Co_2 tension.
Gradual return to normal Cardio-Vascular dynamics.
Lessened Acidaemia
Less distension of cerebral vessels.
Less tendency to sudden over-distension of Vessels.
Relief of Headache.

Preliminary Conclusions of Occipital Headache in Chronic Nephritis:

(1) Too few cases were present in this series to form any definite conclusions.

(2) Occipital headache is not a common symptom in patients with chronic glomerulo-nephritis (2 out of 10 i.e. 20%).

(3) It usually occurs at any time but is precipitated by sudden movement such as rising from bed.

(4) It is always throbbing in character.

(5) Relief is difficult to obtain by drugs. Relief is usually obtained by dietetic treatment of the nephritic state or of the associated left ventricular failure.

(6) It would appear that renal failure predisposes to the cerebral vaso-dilatation and that alteration in the cardiac output suddenly produced, precipitates an attack of headache.

(7) The severity of the headache is associated with renal function, cardiac decompensation, the levels of blood pressure and the degree of acidaemia.

(8) The prognosis of occipital headache is difficult to determine. If due to renal failure alone, the prognosis is bad (Case No.22). If associated with frontal headache and cardiac failure, the prognosis depends upon the degree of recovery from the cardiac failure. If recovery is made from the cardiac failure and the headache improves, the immediate prognosis is good, although the ultimate outcome is the same due to the renal impairment.

Percentage of Relief of Headache in Chronic Glomerulo-Nephritis in this series. (Frontal and Occipital).

(A) Rest and Diet : This gave relief in 40% in this series.

(B) Aspirin : This gave only slight relief in 20%.

(C) Sodium Amytal and Aspirin: This gave no relief in 20%.

(D) Multiple Medication: This gave no relief in 20% of cases.

Other measures employed in the relief of Headache.

Kempner's rice diet which is reputed to give such symptomatic relief in renal and hypertensive vascular disease in 70% of cases, was not employed in those cases of Chronic Glomerulo-nephritis.

Predilection of Site of the Headache.

When the mechanisms of headache production are compared in frontal and occipital headache, it is found that the two mechanisms are identical.

The acidosis of the renal impairment together with the acidosis resulting from cardiac inefficiency, and both being exaggerated by sleep, predispose to the headache by causing cerebral arteriolar dilatation.

The precipicating factor is seen to be sudden movement. Such as rising from bed which suddenly alters the vascular dynamics, resulting in sudden increased blood pressure and cardiac output. This produces sudden over-distension of the cerebral arterioles with resulting headache by the mechanism previously described (Page 106).

The improved blood supply to the kidney, resulting from the increased cardiac output, on rising improves the kidney function, if not grossly impaired, and thus lessens the degree of acidaemia with resulting vaso-constriction of the cerebral arterioles. The duration of the headache, therefore, acts as a prognostic sign as to renal function. When the headache is persistent, in spite of therapy or occurs at any time, the renal function is failing and the patients die of Uraemia.

When such mechanisms are common to both types of headache, it is difficult to explain why in some patients the headache is frontal and in some occipital.

Other predisposing factors, therefore, must determine the site of the headache.

The severity of the frontal headache would appear to be related to the height of the blood pressure, (Diagrams XXXIV. & /XXXV. /Vascular changes as revealed by fundal changes (diagrams XXXVI. the cardiac decompensation (diagrams XXXVII. the renal function (diagrams XXXVIII & the blood Co_2 combining power (diagrams XXXIX).

Severity of the occipital headache would appear to be related to the height of the blood pressure (diagrams XLI & XLI[A] the cardiac decompensation (diagrams XLIII.) the renal function (diagrams XLIV) and the degree of acidaemia (diagrams XLV.)
The condition of the arterioles as suggested by retinal changes does not appear to be associated with the severity of the occipital headache (diagrams XLII).

This suggests, therefore, that in respect of the severity of the headache, vascular factors play some part in frontal headache but none in occipital headache.

The following Table XXVIII also shows :

Headache in Chronic Glomerulo-Nephritis.

Table XXVIII.

	Frontal.	Occipital.
(1) Incidence of headache in total cases	80%	20%
(2) Average age	52 years	42 years.
(3) Sex-Ratio	5 M : 3 F.	1 M : 1 F.
(4) Average weight	10 st. 4 lbs.	9 st. 10 lbs.
(5) Habit: Smoking	4 out of 8 - 50%	1 out of 2 - 50%
Alcoholic	3 " of 8 - 38%	1 " of 2 - 50%
(6) Nerve Supply of area of headache.	Ophthalmic branch of the 5th Cranial Nerve.	(i) Cervical 1 - 3. (ii) Lesser occipital nerve. (iii) Greater occipital nerve.
(7) Acidaemia causing dilatation of cerebral arterioles	(i) Renal failure and Secondary cardiac inefficiency. (ii) Sleep.	(i) Renal failure and Secondary cardiac inefficiency. (ii) Sleep.
(8) Precipitating factors	Sudden movement resulting in sudden increased cardiac output.	Sudden movement resulting in sudden increased cardiac output.
(9) Relationship of Severity:		
(i) Systolic Pressure	R.	R.
(ii) Diastolic pressure	R.	R.
(iii) Fundal changes	R.	N.
(iv) Cardiac decompensation	R.	R.
(v) Blood Urea N_2	R.	R.
(vi) Co_2 combining power	R.	R.
(10) Relationship between (i) Blood Urea N_2 and B.P.	N (R when over 100 mgm%)	R.
(ii) B.P. amd Fundi	N	N
(iii) Fundi and Blood Urea N_2	R	R
(11) Relief:- Rest and diet	+	No relief.
Aspirin	Slight relief	Slight relief.
Sodium Amytal and Aspirin	No relief	No relief.
Multiple Medication	No relief	No relief
(12) Mortality	37% Cause of death - Uraemia, Pericarditis and terminal Broncho Pneumonia	50% Cause of death Uraemia.

Key: R = Relationship; N = No relationship; + = Relief.

Comparison of predisposing and precipitating factors.

Table XXVIII shows the predisposing and precipitating factors in both frontal and occipital headache.

In the predisposing factor, the difference between the two groups are:-

(1) Patients are older who suffer from frontal headache.

(2) More males suffer from frontal headache.

(3) The patients with frontal headache are heavier.

(4) There is no difference between the grpups where the habits of smoking and alcohol are concerned. The effects of which have been already described.

(5) It has been clearly seen that the main predisposing factor in both groups is acidaemia producing vaso-dilatation. This acidaemia results from cardiac decompensation causing an increase of Co_2 in the blood, renal impairment with retention of other acid radials and the physiological acidaemia of sleep. The cases in this series have shown that renal factors, although present in both groups, are of greater severity in the group with occipital headache. While the cardiovascular factor, again present in both groups, are more prevalent in the group suffering from frontal headache.

The renal failure in the group with occipital headache is much more severe than in the group with frontal headache and therefore the acidaemia in the occipital headache is far more profound.

This is in accord with the findings in the group with essential hypertension, where frontal headache is mainly dependent upon cardiac insufficiency and occipital headache is mainly dependent upon renal impairment.

The precipitating factors in both groups is the same, namely a sudden over-distension of cerebral arterioles, already dilated.

Just as in the case of the headache in essential hypertensive patients, why should in some cases the headache be frontal and in some be occipital ? One mechanism suggested in the discussion on essential hypertension was a differential response of the cerebral arterioles to the same stimulus, namely acidaemia, this stimulus, however, varying in nature and intensity.

Another mechanism which receives support from other workers is that the headache will be localized to areas already the seat of some minor lesion such as cervical fibrositis or osteo-arthritis. In this group with chronic nephritis,the younger age group of the patients with occipital headache would not suggest that cervical osteo-arthritis was a cause in these cases. In the patients who recovered,examination of the cervical spine showed no abnormality.

Eye-strain suggested by some workers, as a factor explaining the localization of the headache, will not apply in this series; for in the group with occipital headache, therefore, dependent upon spasm of the frontalis muscle in these cases would surely be situated frontally.

Prognosis and Conclusions in patients with headache, suffering from Chronic Glomerulo-nephritis.

Clinical observations and analysis of patients with headache in Chronic Glomerulo-nephritis, who suffer from headache show that:

(1) The headache is of two types; (a) frontal, and (b) occipital.

(2) Frontal headache occurred in 80% of these cases.

(3) Occipital headache was less frequent. It occurred in 20% of this series.

(4) The character of the headache was the same in both groups, it occurred on wakening or rising from bed in the majority of cases. In a few cases, it was present throughout the day but was worse on rising from bed.

(5) In all cases, it was throbbing in nature.

(6) The duration of the attack was from half an hour to several hours, but tended to last longer when it was situated occipitally. This is similar to the findings in the group with essential hypertension.

(7) Relief could be obtained in only a few cases by drugs in the group with frontal headache. In occipital headache, relief is difficult to obtain by drugs but can be achieved in some cases by the treatment of the nephritic state or of the associated left ventricular failure.

(8) In both groups of cases, just as in the group with essential hypertension, our investigations have shown that the precipitating factors in the production of frontal or occipital headache is the sudden over-distension of dilated cerebral arterioles. This over-distension results from a sudden increase in cardiac output and level of blood pressure produced by rising on wakening.

(8A) Observations on these patients and their response to the many therapeutic measures used, show that a gradual increase in the cardiac output with a resulting gradual elevation of the blood pressure does not produce headache.

(9) The cerebral arteriolar dilatation results from acidaemia. In the group with frontal headache, the acidaemia is caused by cardiac failure ; renal impairment and by sleep. In this group, the acidaemia is mainly due to cardiac failure with excess of Co_2 in the blood. In the group with the occipital headache, the acidaemia results from renal failure, cardiac failure and sleep. In this group, however, the acidaemia is mainly due to renal failure with excess of acid radicles in the blood.

(10) The severity of the frontal headache,although dependent upon renal impairment, in this series of cases bears a direct relationship to the degree of cardiac decompensation, while the severity of the occipital headache is dependent more on renal failure, although cardiac decompensation also plays a part. The severity of the headache, therefore, if situated frontally, is an indication of the degree of cardiac failure complicating the chronic nephritis, while the severity of the occipital headache is an indication only of the degree of renal failure.

(11) Comparison of the many factors present in both groups of cases, show that patients with frontal headache are both older and heavier than are patients with occipital headache. Furthermore, more males suffer from frontal headache than of an occipital headache.
Unlike the findings in essential hypertension, alcoholism and smoking have a similar incidence in both groups. The significance of the sex, and age incidence and the difference in weight are not apparent as aetiological factors.

(12) The explanation of the different sites of headache in these patients is a differential degree of vaso-dilatation of the cerebral arterioles. In frontal headache the dilatation is due to acidaemia of Co_2 retention, secondary to cardiac failure plus the renal acidaemia of renal impairment. This affects the vessels above the tentorium cerebelli and is thus felt in the distribution of the ophthalmic division of the fifth cranial nerve.

In occipital headache, the dilatation is due to the acidaemia of severe renal failure where the acid radicles are the main substances producing acidosis. These affect the vessels below the tentorium cerebelli and the pain is thus felt in the distribution of the first, second and third cervical nerves.

As in the hypertensive group another explanation would be, that the acidaemia, whatever the cause, produces the vaso-dilatation and that the pain from this, is experienced in areas of the scalp where the pain threshold is already lowered by the pre-existing or concomittant disease, such as cervical osteo-arthritis or fibrositis. In this series, however, there was no evidence of such a lesion.

(13) Mortality in these cases was 37% in patients with frontal headache, the patients dying from uraemia plus cardiac failure. In patients with occipital headache the mortality was 50%, the cause of death being uraemia.

3. Chronic Pyelonephritis.

Previously attacks of acute or even chronic pyelitis was regarded by physicians as of no significance except for the possible events of predisposing to stone formation or in rare instances to pyonephrosis. Experience has taught us how wrong this view was. Chronic pyelonephritis is a very serious condition, as it predisposes to progressive and severe hypertension. The chronic inflammatory tissue with resulting fibrositis produces an ischaemic kidney which results in hypertension. (Goldblatt).

It is known that, if the condition be unilateral, then surgical removal will cure the disease and prevent the sequel of hypertension. With the advent of the many and varied anti-biotics at present available, the incidence of chronic pyelonephritis with its hypertension, met with in middle-aged women who had repeated attacks of acute "Inflammation of the Kidneys", so lightly dismissed by their doctors should never now be encountered.

In this series, there was only one patient with chronic pyelonephritis with associated hypertension.

She was a woman aged 44, married with no children. Her present complaint was haematuria and pain in the right loin for seven weeks. Previously ten years ago, she suffered from left-sided renal colic and was found to have a left-sided hydronephrosis. This apparently was considered of no importance to warrant any treatment. During this time, she has been passing increased amounts of urine, which as mentioned earlier for the past seven weeks has been blood-stained.

For two months prior to her admission to hospital, she has had throbbing frontal headaches, worse on rising in the morning and lasting for several hours. Her eye-sight has been poor for the last nine weeks.

Twelve years ago, she had a hysterectomy for uterine fibroids.

On examination, the patient was pale, thin and looked ill. She had deep laboured breathing and had a temperature of 100°F. Her P.B. was $\frac{230}{150}$, the pulse was regular, rate 82 per minute and was of high tension. The heart was enlarged but both sounds were pure. Basal crepitations were present over both lungs.

The urinary output was 2000 c.c., specific gravity 1010, albumin blood and pus were present and culture yielded a growth of B.coli. The blood urea nitrogen on admission was 28 mgm%., but this rose rapidly to 80 mgm before death.

Cystoscopic examination and retrograde pyelography showed a cystitis and deformities of the calyces indicative of bilateral old fibrosis. Fundus examination showed grade III. changes.

The patient was treated as best as she could be with rest and "Sulpha triad" (Sulphathiazole, Sulphadiazine and Sulphamethazine) and the only relief from headaches could be obtained by the administration of morphine grain $\frac{1}{4}$ subcutaneously.

The patient died as a typical case of uraemia.

Post-Mortem showed :-

(1) A congenital hypoplastic left kidney with hydronephrosis and hydrouneter.

(2) Bilateral pyelonephritis.

(3) Arteriosclerosis.

(4) Hypertensive heart.

Discussion as to the production of Headache.

This patient died from uraemia, resulting from the bilateral pyelonephritis. She had a gross diastolic hypertension with fundal changes and had all the characteristic features of the headache described earlier in these cases of hypertension and chronic glomerulo-nephritis.

As in these cases, the main factors are:-

(1) The renal failure producing acidosis.

(2) The cardiac hypertrophy with possible mild inefficiency and increased Co_2 tension in the blood. Both these features resulting in cerebral arteriolar dilatation.

(3) Toxaemia was also a feature and according to Symond may predispose to headache.

As before, the precipitating factors were sudden movement, such as rising from bed, with sudden increased cardiac output, sudden elevation of systolic pressure and a sudden increased force distending the already dilated cerebral arterioles. This as mentioned earlier (pages 106, 139 & 225), produces headache.

An other factor would be spasm of the frontalis muscle due to the eye-strain of the associated changes in the fundus oculi (grade III.).

The duration of the attack supports the theory (pages 229 and 231) that with the improved cardiac output on movement, there is an improved blood supply to the kidneys and if the kidneys are not damaged, the

acidosis is less and vaso-constriction results. In this case (as in the other cases nos. 18, 23, 24.) there was gross renal impairment and the duration of the attack indicated such a condition - the headache lasting several hours.

The changes can be seen in diagrams (a) and (b)

Diagram: A:-

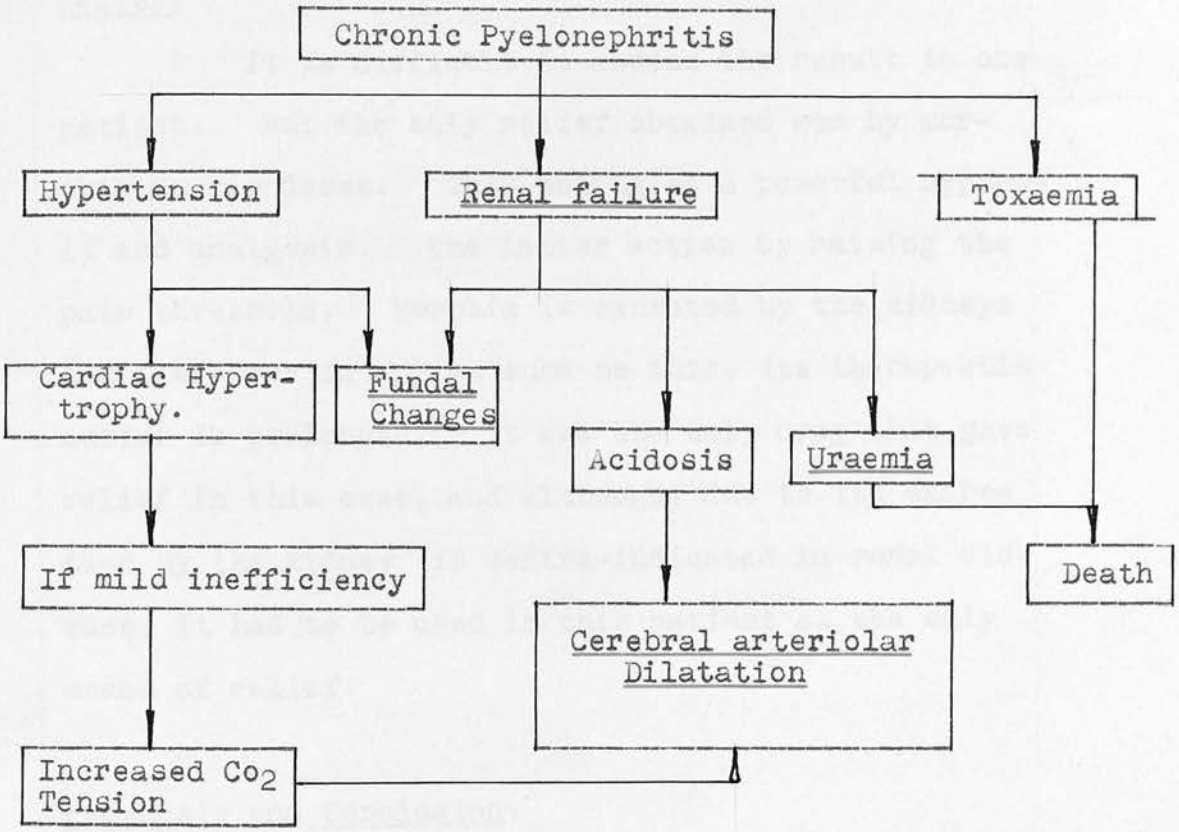
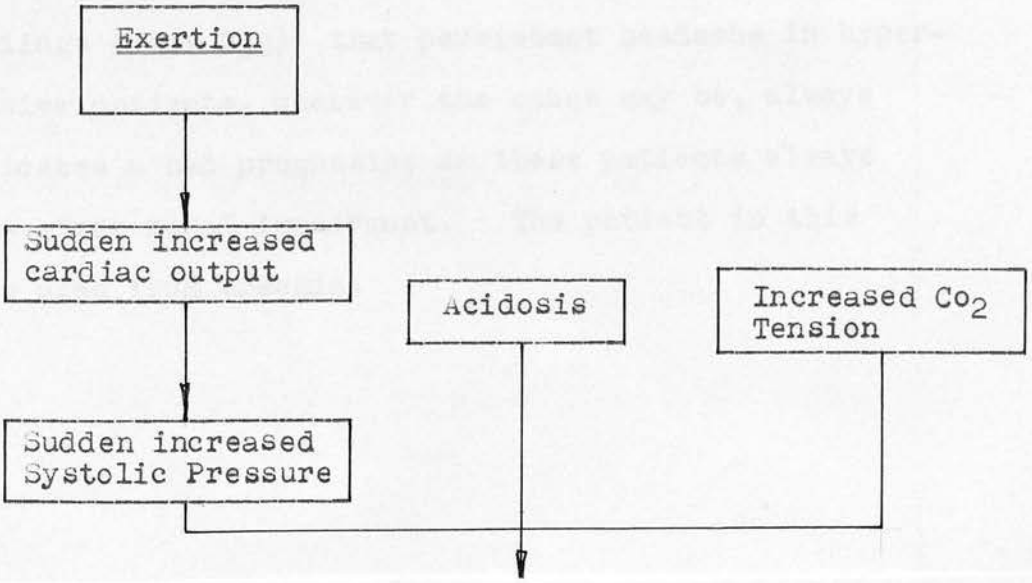


Diagram B:-



Relief:

It is difficult to assess the result in one patient. But the only relief obtained was by morphia in big doses. This acting as a powerful hypnotic and analgesic. The latter action by raising the pain threshold. Morphia is excreted by the kidneys and therefore in a case such as this, its therapeutic action is prolonged. It was the only drug that gave relief in this case, and although, due to its excretion by the kidney is contra-indicated in renal disease, it had to be used in this patient as the only means of relief.

Prognosis and Conclusion:

No conclusions should be made from one case in any series. This case, however, does supply the findings (page 233) that persistant headache in hypertensive patients, whatever the cause may be, always indicates a bad prognosis, as these patients always show gross renal impairment. The patient in this case died from uraemia.

Amyloidosis.

It is well known, that the Amyoidosis complicating chronic septic states may produce the amyloid contracted kidney. This condition results in renal insufficiency and the patient indeed may die of uraemia. Hypertension of the diastolic type is a feature of "amyloid contracted kidney". The mechanism of production of this hypertension is just the same as is the hypertension of chronic glomerulo-nephritis.

The effects of this diastolic hypertension are just the same as the effects of diastolic hypertension in other conditions viz:- effects on the heart, blood vessels, kidneys and other organs. In addition, however, the patient with amyloidosis shows also, a more severe degree of renal impairment due to the amyloid contracted kidney; is usually anaemic due to the effects of the amyloid infiltration of the many organs particularly, the liver and spleen and will also show all the effects of prolonged sepsis.

The three cases of amyloidosis are analysed in Table XXIX.

Analysis of Cases of Amyloidosis.

Table XXIX.

Sex	Female.	Male.	Female.
Age	25	28	50
Weight	-	8 st. 3 lbs.	6 st. 8 lbs.
Habit: Smoking	-	-	-
Alcoholic	-	-	-
Family History	Not relevant.	Not relevant.	Not relevant.
Previous Illness	Pneumonia.	Pneumonia.	Rheumatoid Arthritis.
Presenting Symptoms	Diarrhoea and Vomiting 2 weeks.	Dyspnoea on exertion and constant coughing - 8 mths.	Epigastric pain and Hemetemesis - 5 hrs.
Headache	+	-	+
Duration	2 weeks	-	3 weeks
Site	F	-	F
Character	T	-	T
Onset	Worse on rising	-	Worse on rising
Severity	S	-	M
Duration of Attack	About 1 hour	-	About ½ hour
Precp. factors	Coughing and Vomiting	-	Movements.
Associated Symptoms	-	-	-
Relief	Rest and Sedative Linctus		Rest, and Iron Therapy.
	A		
Pulse Rate	130	100 90	100 80
B.P.	A		
Systolic	150	160 160	90 150
Diastolic	100	100 100	60 100
Heart	Clinically N.	N.	Clinically N.
E.C.G.	N.	N.	L.A.D.
X-Ray of Chest	Left-sided Pl.Effusion Bil. Bronchiestasis	Bilateral Bronchiestasis	-
Abdominal Examination	Liver ++ Spleen++	Liver + Spleen +	Liver ++ Spleen ++
Fundal Changes	III.	N.	N.
Blood Urea Nitrogen	50 mgm%	60 mgm%	24 mgm%
Wassermann Reaction	Negative	Negative	Negative
Congo-red test	Not done	+	+
B.S.R.	45 mm. in 1 hr.	50 mm. in 1 hr.	42 mm. in 1 hr.
Sputum	T.B. Negative	T.B. Negative	-
Urine : Reaction	Acid	Acid	Acid
sp. gr.	1015	1014	1020
Albumin	+	+	+
Blood	-	-	-
Casts	+	+	-
Diagnosis	Bilateral Bronchiectasis and Amyloidosis	Bilateral Bronchiectasis and Amyloidosis	Rheumatoid Arthritis Amyloidosis and Duodenal Ulcer.
Treatment	Rest, Sedation and Kaolin.	Rest, Postural drainage, Penicillin Liver extract and Iron.	Rest. Blood Transfusion Sedation and ulcer diet.
Result	Died.	Improvement.	Improvement.

Key:- F = Frontal. T + Throbbing. S = Severe. N = Normal.

The Summary of these findings are enumerated below :-

Summary of Cases of Amyloidosis:

Total number of cases 3.

A. Cases with Headache:

1.	Incident	-	2 out of 3 cases i.e. 66%.
2.	Sex	-	all females.
3.	Average Age	-	37½ years.
4.	Average Weight	-	6 st. 8 lbs.
5.	Site of Headache	-	Frontal in both cases.
6.	Character	-	Throbbing and worse on rising from bed.
7.	Average Duration of headache	-	2 weeks - 3 weeks.
8.	Average duration of Attack	-	¾ hour.
9.	Precipitating Factors	-	In one case coughing and vomiting.was precipitating factor and in the other anaemic state.
10.	Relief	-	The headache was eased with rest and sedative linctus in the former, and with iron therapy for the anaemia in the latter case.
11.	Average B.P. on Admission	-	150/100
	on Discharge	-	150/100
	On Admission	-	In the latter case B.P. was 90/60 following massive haemetemesis.
12.	Average Blood Urea Nitrogen	-	37 mgm%. Both cases showed evidence of renal impairment.
13.	Causes of Amyloidosis	-	Bilateral bronchiectesis in one case and Rheumatoid Arthritis in another.
14.	Average B.S.R.	-	44 mm. in one hour (Westergren)

B. Case without headache:

1.	Incident	-	1 out of 3 cases i.e. 33%.
2.	Sex	-	Male.
3.	Age	-	28 years.
4.	Weight	-	8 st. 3 lbs.
5.	Average B.P. on Admission	-	160/100
	" Discharge	-	160/100
6.	Blood Urea N_2	-	60 mgm%.
7.	B.S.R.	-	50 mm. in 1 hour. (Westergren).

If these two groups are now compared, it can be seen that :-

	Headache	No Headache.
Incident	66%	33%
Sex	All females	Male
Average age	37½ years	28
Average weight	6 st. 8 lbs.	8 st. 3 lbs.
Average B.P.	150/100	160/100
Average Blood Urea N_2	37 mgm%	60 mgm%
Average B.S.R.	44 mm. in 1 hr.	50 mm. in 1 hr.

Preliminary Conclusion :-

1. The cause of headache in Amyloidosis would appear to be associated with the hypertension and renal involvement and not per se to the amyloid infiltration.

2. The patient without headache, however, has a high B.P. and a higher blood urea nitrogen than the two cases with headache.

3. Some other factors would thus appear to be present to cause the headache. These may be :-
   (1) Severe Anaemia.
   (2) Attacks of retching and vomiting.
   (3) Some unknown factors.

4. One case with headache died of Uraemia and terminally complained also of occipital headache.

5. The Uraemia may have been the deciding factor in the causation of this patient's headache; for, the patient with bronchiectasis had no headache, was not uraemic, and improved sufficiently to be discharged home.

Discussion of production of headache in patients suffering from Amyloidosis:-

One patient, a girl of 25 years, had amyloidosis secondary to bilateral bronchiectasis (Case No. 28). She died from Uraemia. Her presenting symptoms were diarrhoea and vomiting and she also complained of a

throbbing frontal headache which was precipitated by coughing and retching.

The predisposing factors for this girl's Headache would appear to be her amyloidosis, and renal insufficiency and perhaps toxaemia.

The precipitating factors are exertion, such as retching or a paroxysm of coughing.

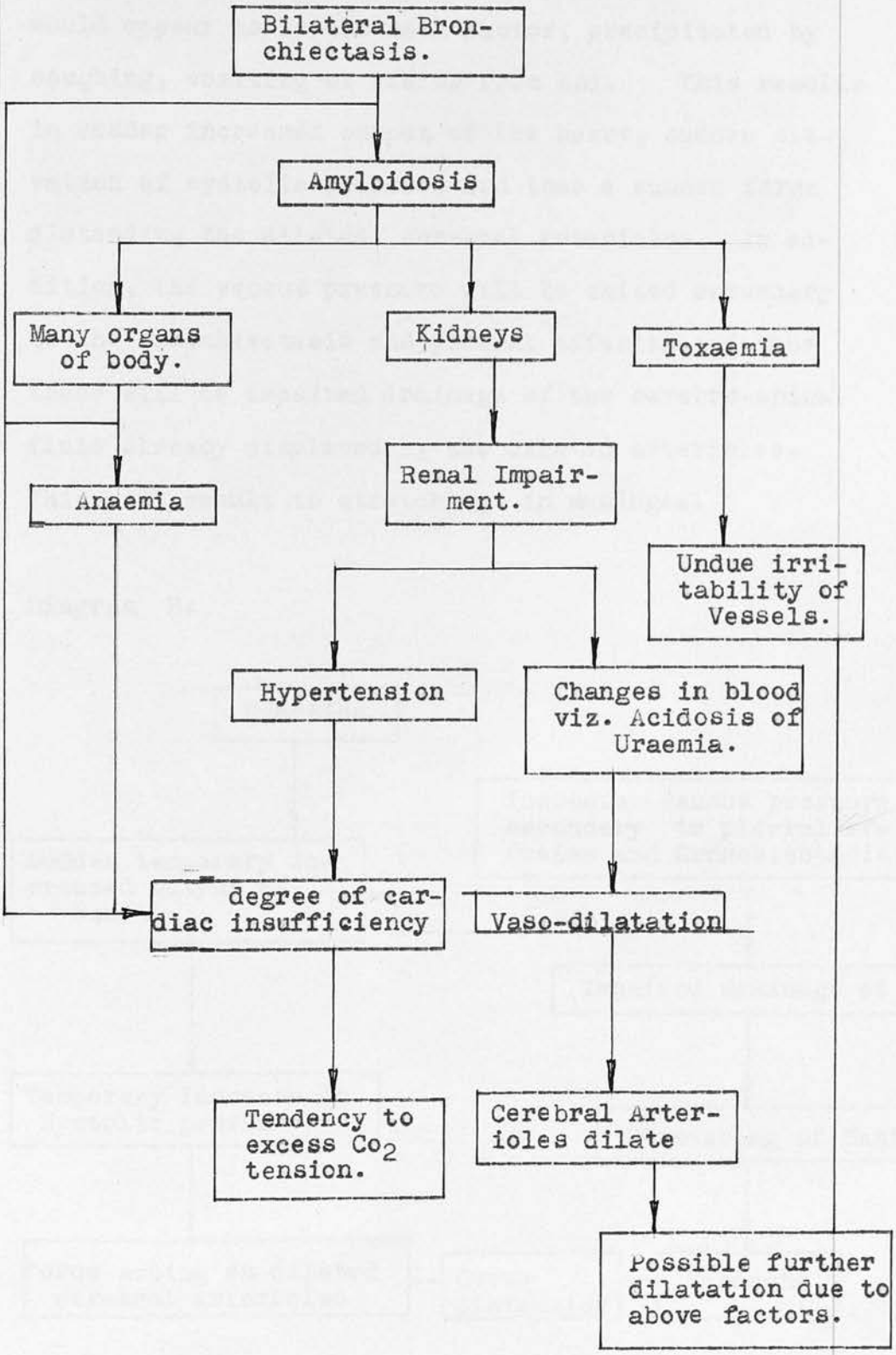
The sequence of events would appear to be Bronchiectasis, producing toxaemia, amyloidosis and cardiac inefficiency.

The Toxaemia :-I would suggest, results in undue irritability of the blood vessels including the cerebral arterioles. (Symond).

Anyloidosis as mentioned earlier, results in renal impairment and hypertension. The renal impairment produces a state of acidosis and uraemia (from which the patient died) with cerebral arteriolar dilatation. Further, the hypertension adds to a heart already inefficient from the effects of bronchiectasis, amyloidosis and anaemia, resulting in further cardiac inefficiency with excess Co_2 tension in the blood and further cerebral arteriolar dilatation.

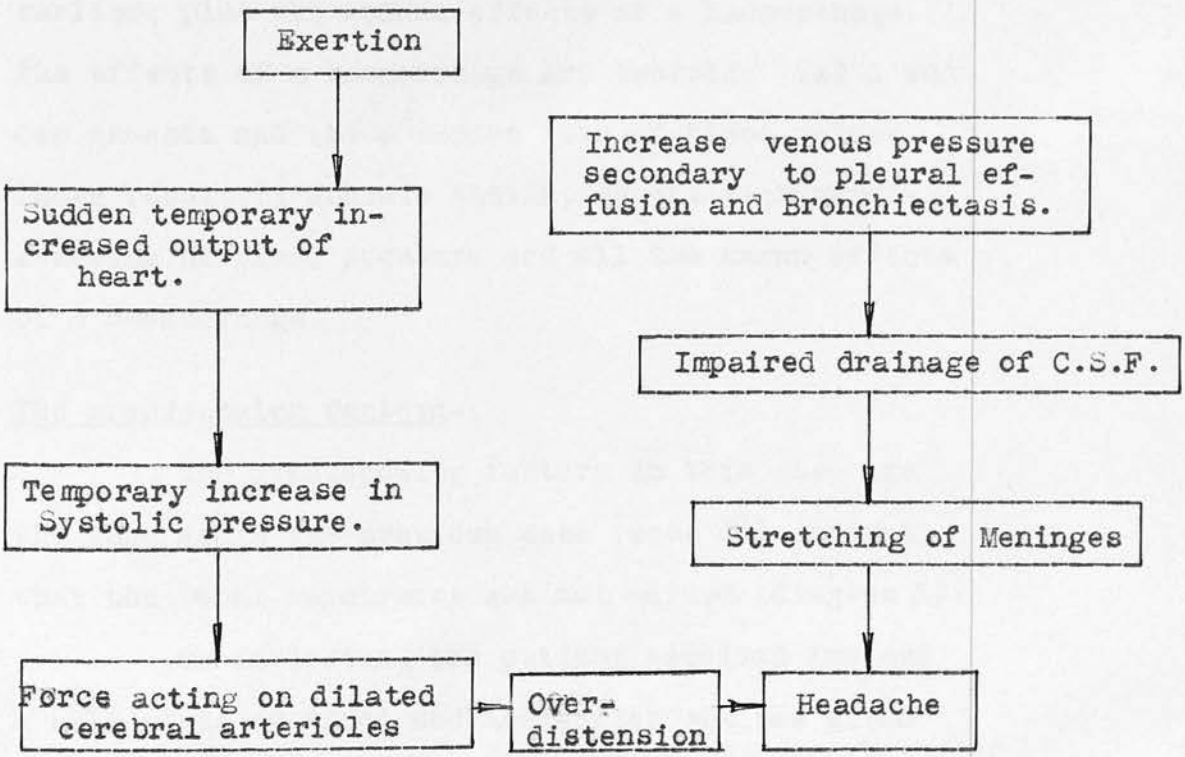
The stage is therefore set for development of headache (Diagram A).

Diagram A:


Bilateral Bronchiectasis.
Amyloidosis
Many organs of body.
Kidneys
Toxaemia
Anaemia
Renal Impairment.
Undue irritability of Vessels.
Hypertension
Changes in blood viz. Acidosis of Uraemia.
degree of cardiac insufficiency
Vaso-dilatation
Tendency to excess Co_2 tension.
Cerebral Arterioles dilate
Possible further dilatation due to above factors.

Precipitating Factors: In this patient, exertion would appear to be the main factor, precipitated by coughing, vomiting or rising from bed. This results in sudden increased output of the heart, sudden elevation of systolic pressure and thus a sudden force distending the dilated cerebral arterioles. In addition, the venous pressure will be raised secondary to the bronchiectasis and pleural effusion and thus there will be impaired drainage of the cerebro-spinal fluid already displaced by the dilated arterioles. This will result in stretching in meninges.

Diagram B:



Before the patient died of Uraemia, she complained of occipital headache, constant and dull in character. This headache must be associated with her uraemic state and its mechanism of production is therefore, the same as the occipital headache encountered in the cases of chronic glomerulo-nephritis who died from uraemia described previously.

In the other case (Case 27), the Amyloidosis was a complication of chronic rheumatoid arthritis. In addition, the patient had a peptic ulcer from which she suffered a haemetemesis. In this patient, therefore, one is dealing with the changes in the body secondary to amyloidosis as mentioned earlier, plus the sudden effects of a haemorrhage. The effects of a haemorrhage are twofold: (a) A sudden anaemia and (b) a sudden loss of blood volume. These result in anaemic anoxia, shock, tachycardia, lowering of blood pressure and all the known effects of a haemorrhage.

The predisposing factors-

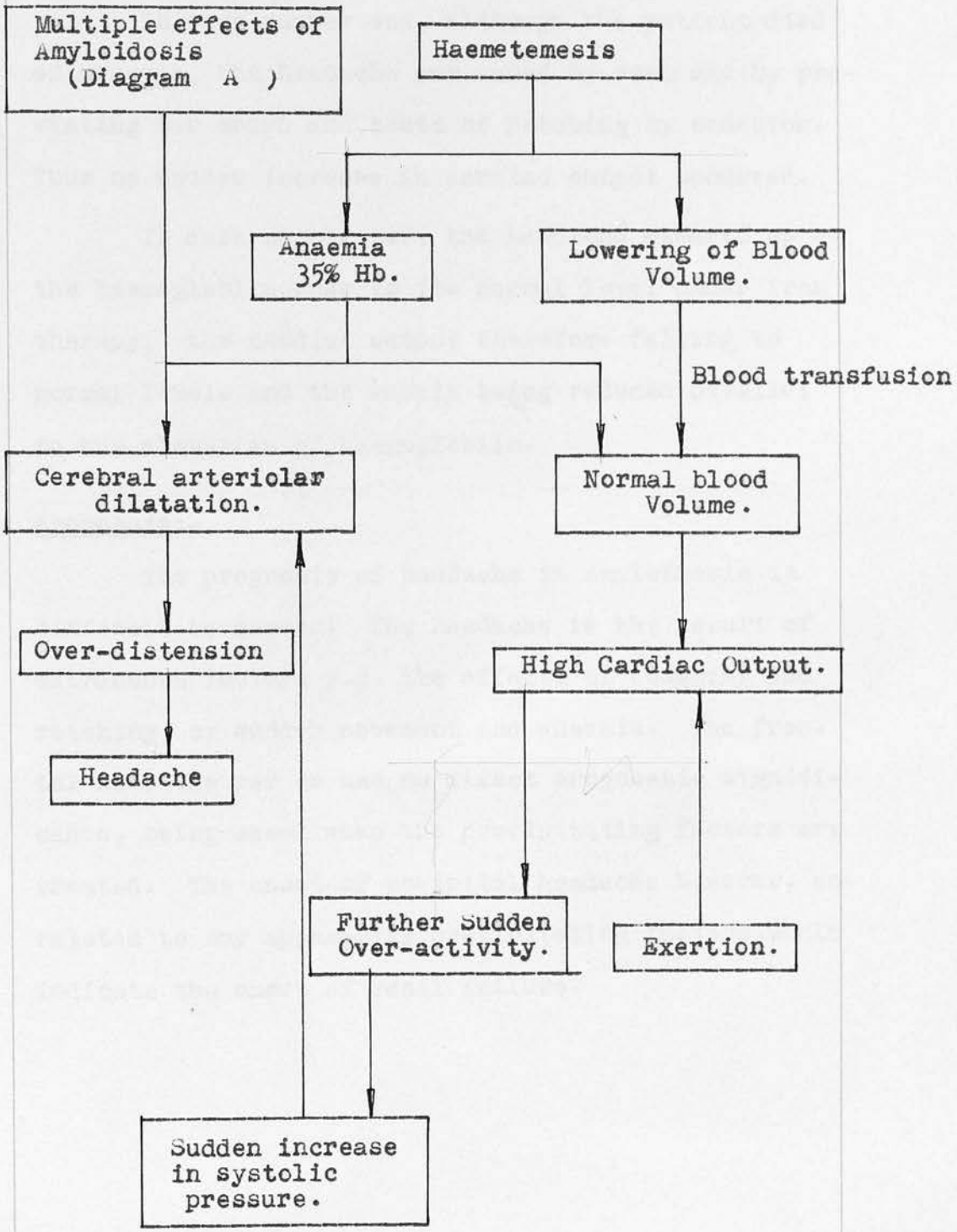
The predisposing factors in this case are the same as in the previous case (case 28) except that the renal impairment was not marked (diagram A).

On admission, the patient received two and a half pints of blood and thereafter she was given ferrous sulphate in addition to the treatment of her peptic ulcer. It was during the period of this treatment (when the haemoglobin after transfusion was 35%)

that she complained of headache, worse on raising the head. The blood volume at this period is normal, thus the effects of shock and pre-renal azotaemia, although present initially do not play any part at this time.

One is dealing therefore with the effects of anaemia plus sudden movement. Sharpey-Schafer(1944) shows that anaemia with a normal blood volume results in high output failure i.e. a state of a hyperkinetic cardiac activity with an increased systolic pressure. Sudden movement will increase this output to a higher degree, the systolic pressure will be therefore, suddenly raised. This increased force will act upon cerebral arterioles dilated due to the multiple effects of the Amyloidosis (see Diagram A) plus the effects of the anaemia (Hb 35%). This over-distension, it is suggested, will explain the headache in this case.

Diagram C:


Multiple effects of Amyloidosis (Diagram A)
Haemetemesis
Anaemia 35% Hb.
Lowering of Blood Volume.
Blood transfusion
Cerebral arteriolar dilatation.
Normal blood Volume.
Over-distension
High Cardiac Output.
Headache
Further Sudden Over-activity.
Exertion.
Sudden increase in systolic pressure.

Relief of Headache:

In case number one, although the patient died of uraemia, the headache was eased by rest and by preventing her cough and bouts of retching by sedation. Thus no sudden increase in cardiac output occurred.

In case number two, the headache cleared up as the haemogloblin rose to its normal level under iron therapy; the cardiac output therefore falling to normal levels and the anoxia being reduced parallel to the elevation of haemogloblin.

Prognosis:-

The prognosis of headache in Amyloidosis is difficult to assess. The headache is the result of extraneous factors e.g. the effects of coughing and retching or sudden movement and anaemia. The frontal headache per se has no direct prognostic significance, being eased when the precipitating factors are treated. The onset of occipital headache however, unrelated to any apparently precipitating factors, would indicate the onset of renal failure.

5. Subarachnoid Haemorrhage.

Subarachnoid haemorrhage occurs from many causes. It can be defined as the only condition associated with bleeding into subarachnoid space. Of the many causes of this condition, the five cases in this series would suggest that the cause was a ruptured congenital aneurysm or rupture of a vessel, forming the circle of Willis, weakened by atheroma and hypertension. Such a spontaneous subarachnoid haemorrhage produces a sudden elevation of intra-cranial pressure and the presence of the blood in the subarachnoid space irritates the meninges. One is dealing, therefore, with (1) the cause of the haemorrhage, and (2) the effects viz., increased intra-cranial pressure and meningeal irritation.

Table XXX. (page 274) shows the analysis of five cases in this series). (Appendix cases 30 - 34).

Analysis of Cases of Subarachnoid Haemorrhage.

Table XXX.

Sex	Male	Female	Female	Female	Female
Age	25	58	75	56	48
Weight	10 st. 7 lbs.	9 st. 12 lbs.	10 st. 5 lbs.	11 st. 2 lbs.	-
Habit: Smoking	-	-	-	-	-
Alcoholic	-	-	-	-	-
Family History	Not relevant	Mother - Cerebral Haemorrhage Father - Heart Attack	Mother Hypertension	Not known	Not known
Previous Illness	Not relevant	Not relevant	Not relevant	Not relevant	Not relevant
Presenting Symptoms	Sudden frontal headache.	Sudden bursting Headache.	Dyspepsia and Sudden frontal Headache.	Unconscious Headache.	Sudden right Temporal headache.
Headache	+	+	+	+	+
Duration	12 hours.	Several hours.	3 days.	Sudden.	2 days.
Site	F.	"all over"	F	F	Right Temporal.
Character	Intense bursting and progressive	Bursting and Terrible	Bursting	Bursting	Severe and bursting.
Onset	S	S	S	S	S
Precip.factors	Onset of Haemorrhage	Haemorrhage	Haemorrhage	Haemorrhage	Haemorrhage.
Associated Symptoms	V	V and Dizziness	-	V	V
Relief	L.P. and Pethidine	L.P. Sedation	L.P. Sedation	L.P.	-
Temperature	99·4°F.	100°F	97°F	99°F	100°F
Pulse Rate	A 72 D 70	52 80	72 72	40 80	75
B.P.	A D				
Systolic	140 120	180 160	190 180	160 160	150
Diastolic	100 80	90 90	100 100	100 90	100
Heart	N	Clinically E	E and H_2 ++	Clinically E.	N.
Fundus Oculi	N.	N.	I.	N.	N.
C.S.F. Colour	Blood stained	Blood stained	Blood stained	Blood stained	Blood stained
Pressure	200 mm H_2O	200 mm H_2O	300 mm H_2O	320 mm H_2O	250 mgm%
Protein	70 mgm%	80 mgm%	80 mgm%	85 mgm%	150 mgm%
R.B.C.	Loaded	Loaded	Plenty	Plenty	Plenty
Sugar	85 mgm%	80 mgm%	80 mgm%	72 mgm%	76 mgm%
Chloride	680 mgm%	720 mgm%	700 mgm%	710 mgm%	700 mgm%
W.R.	Negative	Negative	Negative	Negative	Negative
Urine	N.	N.	N.	N.	N.
State of the patient	Semi-conscious.	Not unconscious.	Not unconscious.	Unconscious	Fully conscious on Admission.
Etiology	Congenital Aneurysm	Atheromatous Aneurysm ?	Atheromatous Aneurysm ?	Atheromatous Aneurysm ?	Congenital Aneurysm.
Treatment	Rest, L.P. Sedations.	Rest, L.P. Sedation.	Bed Rest, L.P. and Aspirin.	Rest, L.P. Aspirin.	Rest and Diagnostic L.P.
Result	R.	R.	R.	R.	Died.

Key: F = Frontal. S = Sudden. L.P. = Lumbar Puncture. V = Vomiting.
N = Normal. R = Relief.

The Analysis of these cases are summarized as follows:-

Summary of Cases of Subarachnoid Haemorrhage:-

The total number of cases - 5.

A. Cases with Headache.

1. The patient with headache. 5 out of 5 cases i.e. 100%.
2. Sex : 4 Females and one Male.
3. Average age : 52 years.
4. Average Weight: 10 st. 7 lbs.
5. Site of Headache : Frontal in 3 cases; Right temporal in one case, and all over the head in another.
6. Character of Headache : Bursting and severe in all cases.
7. Duration of the Headache : From few hours to 3 days.
8. Associated Symptoms : Three cases suffered from Vomiting and one from dizziness.
9. Relief : Obtained by Lumbar-puncture and also eased slightly by Aspirin and Pethidine.
10. Average B.P. on Admission $\frac{164}{98}$
11. Average B.P. on Discharge - $\frac{155}{90}$.
12. Average C.S.F. Pressure - 254 mm of H_2O
13. Fundal Examination: Stage I. in one case, and Normal in others.

Preliminary Conclusions:

1. Five patients, suffering from subarachnoid haemorrhage.
2. All suffered from severe bursting frontal headache. This was associated with an increase in blood pressure both systolic and diastolic and the great increase in pressure of cerebro-spinal fluid.
3. Three of the patients were elderly and even after recovery showed an elevated blood pressure. These patients were presumably suffering from essential hypertension, even so, the increase in pressure of cerebro-spinal fluid resulting from the haemorrhage, elevated their blood pressure further.

4. Relief was obtained by lumbar-puncture in four cases. (Fifth case waa deeply comatous and died soon after admission.)

5. One can conclude that the headache in these patients was directly related to the sudden increase in C.S.F. pressure.

Discussion as to the Mechanism of Headache Production.

The cases from the start must be divided into two distinct groups:

(A) Where there was a known history of hypertension existing previously (Cases, aged 58,75 and 56).

(B) Where hypertension was a feature of the subarachnoid haemorrhage and blood pressure returned to normal after recovery (Case aged 25).

A. Pre-existing hypertension:

These patients were suffering from essential hypertension, Subarachnoid haemorrhage occurred in these patients and as the ages ranged from 56-75, it must be assumed, the haemorrhage occurred secondary to atheroma and weakening of the arterial wall; for congenital aneurysm to be present at the age of 50 and over is extremely unusual. Many authors have discussed this problem including Osler, Turnbull, Forbes, Glynn, Pitt and Carmichael. The latter author tried to reconcile the views of the many workers and concludes that atheroma may occur in a hitherto unruptured and undiscovered symptomless congenital aneurysm and predisposes this aneurysm to spontaneous rupture.

One is dealing, therefore with the effects on the body of diastolic essential hypertension which has been described at length (pages 21 - 33).

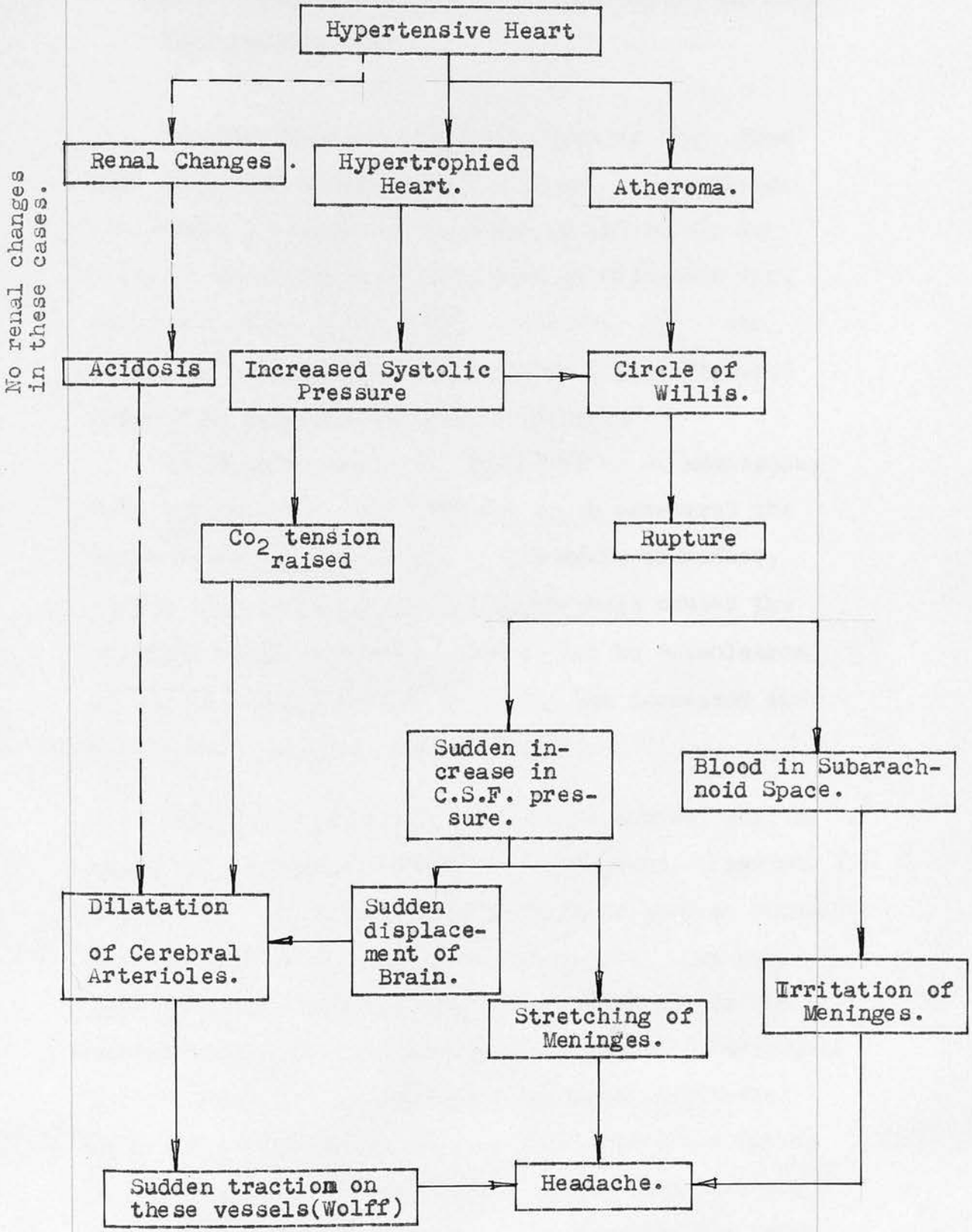
The predisposing factors in the mechanism of headache therefore, are the same, as in that group of essential diastolic hypertensive cases, but would appear to play no part in the headache production in these cases. The spontaneous subarachnoid haemorrhage is the one and only cause of the headache in these three cases, for previous to this, there was no complaint of headache.

The cerebro-spinal fluid pressure in these cases was 200, 300 and 320 mm H_2o. There would therefore be considerable stretching of the meninges with the production of headache. (Brain).

In addition, the presence of the blood would act as an irritant and further increase the headache. Furthermore, the sudden shift of the brain due to the increased intra-cranial pressure would produce traction on the dilated cerebral arterioles and further add to the headache (Wolff).

The Diagram A illustrates this mechanism.

Diagram


Hypertensive Heart
Renal Changes.
Hypertrophied Heart.
Atheroma.
No renal changes in these cases.
Acidosis
Increased Systolic Pressure
Circle of Willis.
Co_2 tension raised
Rupture
Sudden increase in C.S.F. pressure.
Blood in Subarachnoid Space.
Dilatation of Cerebral Arterioles.
Sudden displacement of Brain.
Irritation of Meninges.
Stretching of Meninges.
Sudden traction on these vessels(Wolff)
Headache.

B. Where hypertension was a feature of the Subarachnoid haemorrhage:-

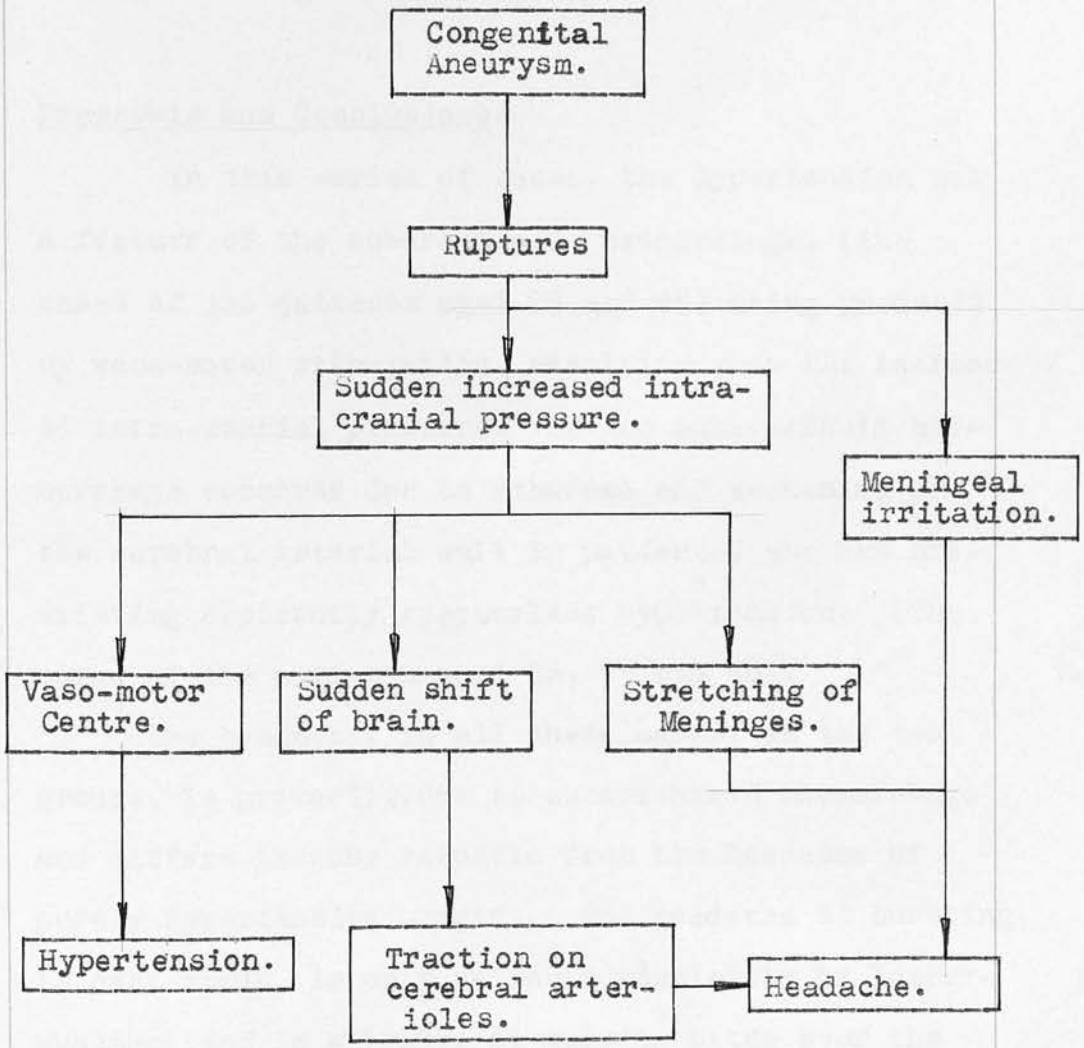
The fourth patient aged 48, (case No. 34) died from increased intra-cranial pressure. A post-mortem showed a congenital aneurysm of the circle of Willis. This case therefore, will be discussed with the fifth case, a boy of 25, (case No. 30) who also had a subarachnoid haemorrhage from a ruptured congenital aneurysm and who recovered.

Both these cases had hypertension on admission, B.P. $\frac{140}{100}$ and $\frac{150}{100}$. In the case which recovered the blood pressure fell to $\frac{120}{80}$. One must, therefore, assume that the subarachnoid haemorrhage caused the elevated blood pressure. This is due to stimulation of the vaso-motor centre caused by the increased intra-cranial pressure (Samson-Wright).

This elevated blood pressure is sudden, and, as shown in the case of the boy aged 25 years, (case No. 30) transient. No pathological effects of such an elevated blood pressure therefore will occur. The headache in these cases therefore, is purely due to the sudden alteration in intra-cranial tension. There will be stretching of the meninges, meningeal irritation and sudden traction on the cerebral arterioles due to the sudden shift of the brain. These three factors as before, result in headache. If the bleeding continued, as in the case of the female aged 48 (see Table No. XXX.) the patient dies of respiratory

and vaso-motor paralysis. When the bleeding stops, either spontaneously as in the case of the boy aged 25, or by surgical means, the intra-cranial pressure falls back to normal, the vaso-motor centre is no longer stimulated and the blood pressure returns to its original pre-haemorrhage level, as in the case in this series, of the boy aged 25 years.

Diagram B:



Relief:

Lumbar puncture relieved the headache in every case. In some patients, the lumbar puncture had to be repeated. Pethidine and Aspirin were used in several patients, they did not relieve the headache much. They presumably acted by raising the pain threshold of the individual.

Lumbar puncture was the only true therapeutic measure which gave complete relief.

Prognosis and Conclusions:

In this series of cases, the hypertension was a feature of the subarachnoid haemorrhage, (the cases of the patients aged 25 and 48) being produced by vaso-motor stimulation, resulting from the increased intra-cranial pressure. Or the subarachnoid haemorrhage occurred due to atheroma and weakening of the cerebral arterial wall in patients, who had pre-existing apparently symptomless hypertension. (The cases of the patients aged 56, 75 and 58).

The headache, in all these cases, in the two groups, is primarily, due to subarachnoid haemorrhage and differs in many respects from the headache of purely hypertensive origin. The headache is bursting, is continuous, is only relieved completely by lumbar-puncture and is situated at varying sites over the

skull. It has therefore, all the features of a headache of increased intra-cranial pressure.

The prognosis therefore, is primarily that of the patient's response to treatment and as to whether there is a recurrence of bleeding. In young patients, associated hypertension should revert to normal levels as recovery occurs. In the older patient suffering from hypertension, the presence of this hypertension may predispose to further bleeding from the original site or from fresh areas of weakened atheromatous vessel walls.

6. Cerebral Tumours:

The presence of a space occupying lesion in the brain results in increased intracranial pressure and the various localizing signs of such a lesion. Unless a haemorrhage occurs in the tumour or there is a sudden onset of hydrocephalus, all the changes occur gradually. The main effects, therefore, result from the slowly progressive increasing intra-cranial pressure.

Table No. XXXI. (page 284) shows the analysis of four cases of cerebral tumours in this series. (Appendix Cases 35, 36, 37 & 38.)

Analysis of Cases of Cerebral Tumours.

Table XXXI.

Sex	Male	Male	Male	Male
Age	49	32	58	48
Weight	7 st. 12½ lbs.	9 st. 13 lbs.	13 st. 4 lbs.	7 st. 7 lbs.
Habit: Smoking	-	-	-	+
Alcoholic	-	-	-	-
Family History	Not relevant	Not relevant	F - Cerebral Haemorrhage M - Cardiac failure	Not relevant
Previous Illness	Not relevant	Not relevant	-	Nil
Presenting Symptoms	Fits preceded by Headache.	Jacksonian fits.	Change in personality - 6 months.	Paralysis of left arm and hand. Weakness of left leg.
Headache	+	+	-	+
Duration	4 weeks.	8 months.	-	4 weeks.
Site	Over left eye	Rt. temporal.	-	Occipital.
Character	Sharp and constant.	Dull and constant.	-	Dull.
Onset	Any time.	Just before fits.	-	Constant.
Duration of Attack	About 1 hour.	½ hour.	-	Constant.
Precip.factors	-	-	-	Worse in morning.
Associated Symptoms.	-	-	-	-
Relief	Relieved after fits and morphine.	Relief after fits.	-	Unrelieved by anything.
Pulse Rate	50	80	90	70
B.P.				
Systolic / Diastolic	170 / 70	170 / 90 — 140 / 80	170 / 100	150 / 110
Heart	Normal.	Normal.	Normal.	Normal.
Fundus Oculi	Bilateral Papilloedema.	Normal	Bilateral Papilloedema.	Bilateral Papilloedema.
X-Ray of Skull	-	Calcification of Vascular markings. Rt. temporal zone.	Normal.	-
Ventriculography	Filling defect in left lateral Ventricle.	-	Both lateral Ventricles dilated. Displaced post. ½ of floor of left lateral ventricles etc.	-
Electro-encephalography	Dysrythmia over left parietal	Localised dysrythmia rt. fronto-temporal zone.	-	
C.S.F. Colour	Clear.	Clear.	Clear.	Clear.
Pressure	100 mm H_2O	120 mm H_2O	200 mm H_2O	150 mm H_2O
Protein	100 mgm%	120 mgm%	80 mgm%	65 mgm%
W.B.C.	3 per c.mm.	4 per c. mm.	Nil	1 per c. mm.
R.B.C.	Nil	Nil	Nil	Nil
Chloride	718 mgm%	716 mgm%	710 mgm%	660 mgm%
Glucose	72 "	69 "	85 "	69 "
W.R.	Negative	Negative	Negative	Negative
Urine	Normal	Normal	Normal	Normal
Diagnosis	Left-sided Cerebral Tumour.	Meningioma.	Cerebral Tumour	Cerebral Tumour.
Treatment	Surgical	Surgically removed.	Surgically attempted.	Symptomatic.
Result	Died.	Cure.	Died.	Died.

They are summarized and salient features of the patients with and without headache are compared.

Summary of cases of Cerebral Tumours:

The total number of Cases 4.

A. Cases with Headache.

1. Incident - 3 out of 4 cases i.e. 75%.
2. Sex - All are males.
3. Average age - 43 years.
4. Average weight - 8 st. 6 lbs.
5. Site of headache - Left Frontal, right temporal and occipital.
6. Character - Dull and constant in three cases, relieved by fits in two cases and by morphia in one of these cases. The third patient obtained no relief.
7. Duration of Headache - 13 weeks.
8. Average Duration of Attack - ½ hour - constant.
9. Average Systolic B.P. on Admission - 125 mm of H_2O.
10. Fundal changes - Bilateral papilloedema in two of the patients with headache and no papilloedema in one patient. This latter patient was found to have meningioma and recovered.
11. The other two patients had glioma and died.

B. Cases without Headache:

1. Incident - One out of 4 cases, i.e. 25%.
2. Sex - Male.
3. Average age - 58 years.
4. Average weight - 13 st. 4 lbs.
5. Average B.P. on Admission - $\frac{170}{100}$
6. Average C.S.F pressure - 200 mm of H_2o.
7. Fundal changes - Bilatteral papilloedema.

If these two groups are now compared, it can be seen that :-

	With Headache.	No Headache.
Incident	75%	25%
Sex	All males	Male
Average Age	43 years	58 years
Average weight	8 st. 6 lbs.	13 st. 4 lbs.
Average B.P. on Admission	163/90	170/100
Average C.S.F. Pressure	125 mm H_2O.	200 mm H_2O.

Preliminary Conclusion:

1. Of the three patients with cerebral tumours, only one patient had diastolic hypertension, and his urine and cardio-vascular system were normal ; one can conclude that his diastolic hypertension was associated with the cerebral tumour.

2. The other two patients had systolic hypertension only. The headache was dull and constant in these cases and was not associated with an increase in C.S. Fluid pressure, as revealed by lumbar puncture.

3. The presence of papilloedema, however, would indicate an increase in the C.S.F. pressure in the skull.

All one can conclude from these cases, is that the cerebral tumours produce a constant headache and in one case was associated with diastolic hypertension.

Discussion as to the Mechanism of Headache Production:

In this group of cases, two patients aged 49 and 32 (Cases No. 35 and 36) suffering from a glioma, which had a fatal termination; and a meningioma which recovered after operation, had an elevated systolic pressure. It is assumed that this elevation of the

systolic pressure, which in these cases was transient, was due to excitement. The levels were $\frac{170}{70}$ and $\frac{170}{90}$, the latter fell to $\frac{140}{80}$.

No further discussion is needed in these cases except to state that the headache was dull and constant and was obviously a headache of meningeal stretching by the increased intra-cranial pressure of the glioma and the presence of the meningioma respectively.

The third patient, a man aged 48 years (case No 38) died from a gliobastoma. The typical constant headache of increased intra-cranial pressure was present in this case, situated occipitally and exaggerated by movement. The blood pressure was $\frac{150}{110}$, there was no history of hypertension previously and at post-mortem no evidence, naked eye or microscopic, of hypertension was found. It is feasible therefore to state that the hypertension was a feature of the cerebral tumour. As in the cases with subarachnoid haemorrhage, the increased intracranial pressure in this case (although the cerebro-spinal fluid pressure was only 150 mm of H_2O, there was bilateral papilloedema) would stimulate the vaso-motor and result in hypertension.

The headache, therefore is due to the tumour, the hypertension being only associated with the increased intra-cranial pressure (as in the patients with subarachnoid haemorrhage). Movement in this

case made the headache worse. One can assume that altered cardiac output would increase the systolic pressure and thus increased the force acting on the cerebral arterioles already distorted by shifting of the brain and by increased intra-cranial pressure.

Diagrams A and B illustrate this :-

A.

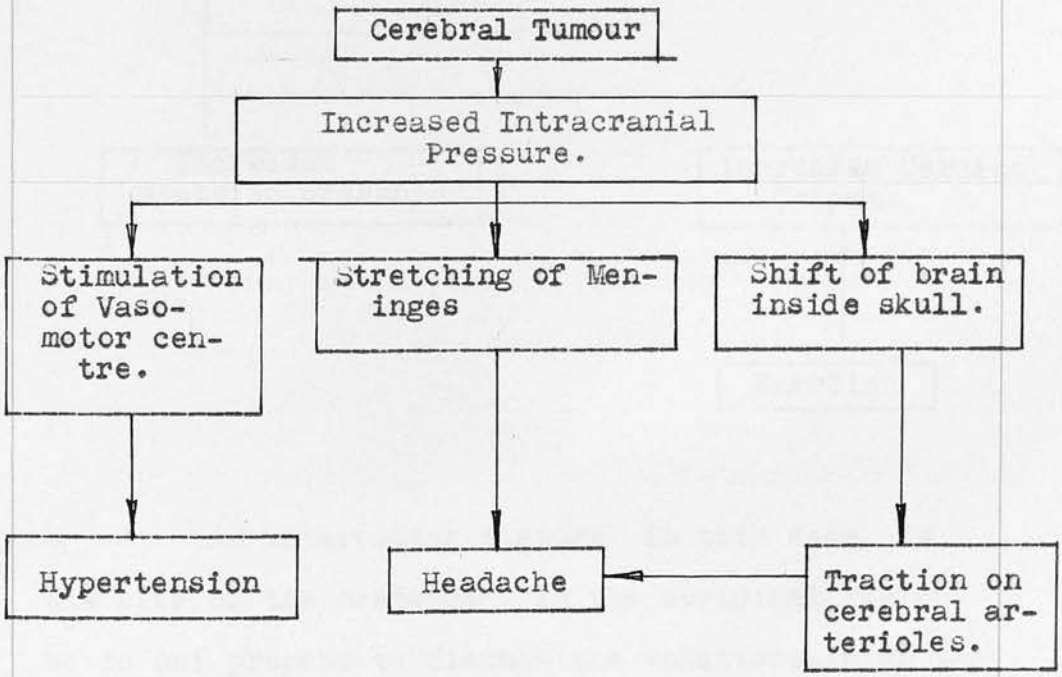
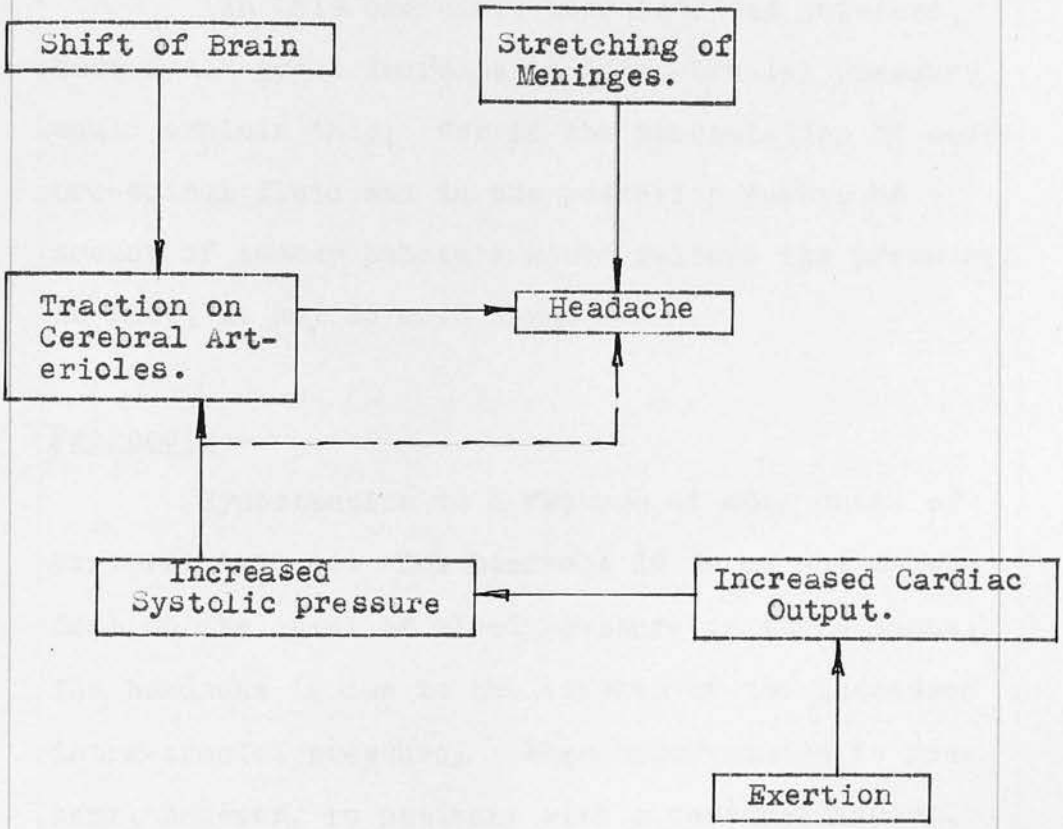


Diagram B



An interesting feature in this case, is the site of the headache - in the occipital region. We do not propose to discuss the relationship of the location of cerebral tumours with the sites of headache, but occipital headache usually means an increased intra-cranial pressure in the posterior fossa of the skull. This is borne out in this patient who had bilateral papilloedema. Yet a normal C.S.F. pressure (150 mm of H_2O), indicating increased posterior fossa pressure with a "coning" of the medulla. Perhaps the vaso-motor centre was stimulated in this way.

Relief:

In this one case, no relief was obtained, here again great increase in intra-cranial pressure would explain this; for if the accumulation of cerebro-spinal fluid was in the posterior fossa, no amount of lumbar puncture would relieve the pressure. In fact, it may do more harm.

Prognosis:-

Hypertension is a feature of some cases of cerebral tumours. The headache is in no way dependent on the level of blood pressure in these cases. The headache is due to the effects of the increased intra-cranial pressure. When hypertension is present, however, in patients with a cerebral tumour, the prognosis is usually grave indicating stimulation of the vaso-motor centre by increased pressure in the posterior fossa with possible "coning of the medulla" leading to sudden death.

7. Cushing's Syndrome.

Harvey Cushing described his typical cases in 1932 and stated, they were due to a basophil adenoma of the pituitary gland. Nowadays, we think that adenoma or hyperplasia of the supra-renal cortex is the primary cause and that the increase in number of basophils with hyaline degeneration in the pituitary is secondary to this, although a similar picture may result from certain ovarian tumours e.g. arrhenoblastoma or thymic tumours.

The typical Cushing's Syndrome picture is well known - the fat beared woman of the circus. Little do the onlookers realize, however, the altered state of these patients' cardio-vascular, endocrine system and their grave prognosis, the patients usually dying from the effects of hypertension.

In Cushing's Syndrome, the important features that concern the subject matter of this thesis are the increased blood volume, the polycythaemia, the excess androgen production, the altered state of other endocrine glands and the many effects of these.

The increased blood volume results in hypertension. The hypertension in time, affects the organs of the body as described earlier.

The polycythaemia alters the viscosity of the blood, tends to make the hypertension worse and predisposes to thrombosis.

The excess androgen production results in an

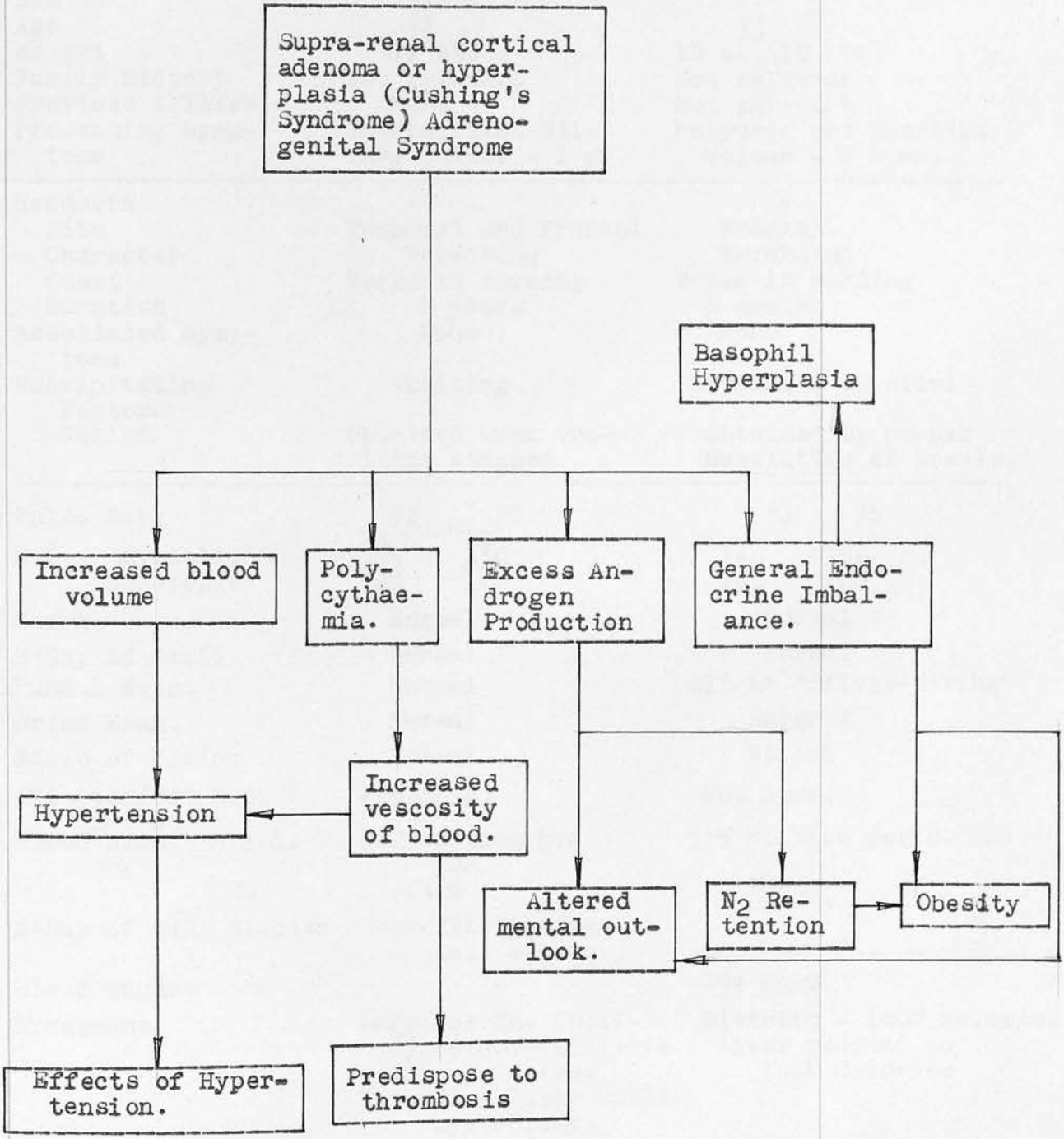
altered mental outlook and conservation of nitrogen leads to a great increase in weight. This latter state worsens the prognosis of the hypertension.

The altered state of the other endocrine glands results in cessation of ovarian function, hyperplasia and hyaline degeneration of the basophils of the pituitary and a general endocrine imbalance. The ultimate result being a further increase in weight and further mental changes.

All these factors play a part in these cases.

These changes can be represented in the following diagram.

Diagram A:



The cases of Cushing's Syndrome are analysed in Table No. XXXII. (Appendix Cases Nos. 39 & 40).

Analysis of Cases of Cushing's Syndrome.

Table XXXII.

Sex	F.	F.
Age	31	33
Weight	17 st.	16 st. 10 lbs.
Family History	Not relevant	Not relevant
Previous Illness	None	Not relevant
Presenting Symptoms	Intermittent Biliary Colics - 1 yr.	Polyuria and Pruritis Vulvae - 4 mths.
Headache	+	+
Site	Temporal and Frontal	Frontal
Character	Throbbing	Throbbing
Onset	Worse in morning	Worse in morning
Duration	8 years	6 months
Associated Symptoms	None	None
Precipitating Factors	Vomiting	Straining at stool
Relief	Obtained when vomiting stopped	Obtained by proper Regulation of Bowels.
Pulse Rate	72 70	82 78
B.P. Systolic (A / D)	145 / 150	140 / 150
Diastolic	100 / 100	100 / 100
Heart	Normal	Normal
X-Ray of Skull	Normal	Normal
Fundal Exam.	Normal	Slight "Silver-wiring"
Urine Exam.	Normal	Sugar +
Field of Vision	Normal	Normal
Ketosteriods Estimation	Not done.	Not done.
Blood Exam. R.B.C.	5·2 million per cmm.	5·5 million per c. mm.
Hb	110%	109%
X-Ray of Gall Bladder	Non-functioning c Gall stones.	-
Blood Sugar	-	194 mgm%
Treatment	Only for Ch. Cholicystitis. Dietetic 1000 Calories. Thyroid - later Cholicystectomy.	Dietetic - 1600 Calories later reduced to 1000 Calories
Result	Symptoms of G.B. disease improved.	Sugar-free; Pruritis Vulvae cleared up. Headache relieved.

Summary of the cases of Cushing's Syndrome:

The total number of cases 2.

A. Castes with headache.

1. Incident - 2 out of 2 cases i.e. 100%
2. Sex - Both females.
3. Average Age - 32 years.
4. Average weight - 16 st. 12 lbs.
5. Site of Headache- Mainly frontal, throbbing in character, worse on rising and precipitated by vomiting and straining at stool.
6. Relief - Obtained by proper regulation of bowel and by stopping vomiting.
7. Average B.P. on admission - $\frac{142}{100}$
8. Average B.P. on discharge - $\frac{150}{100}$
9. Average Hb% - 110%
10. Fundal Changes - Stage I. in one patient, apart from this fact and the elevated B.P., there were no other cardiac-vascular nor renal changes.
11. One patient was a mild diebetic and controlled by diet only.

Preliminary Conclusion:-

1. The headache was similar in character to that associated with the essential hypertension.

2. The headache in Cushing's Syndrome would appear to be associated with the elevated blood pressure, increased blood volume and polycythaemia in these cases.

Discussion as to the production of Headache.

The predisposing factors, in the mechanism of headache in the cases of these two women are the many effects on the body of the adreno-genital Syndrome.

The precipitating factors in these cases are effort or sudden movement.

We have to consider, therefore, these two factors.

(1) Predisposing factors:

The changes in the body resulting from the adreno-genital Syndrome are mainly those of prolonged and profound hypertension, together with polycythaemia, obesity and possibly an altered personality.

In the cases in this series, the main brunt of the hypertension has fallen upon the heart and blood vessels. No evidence was present of renal involvement. The heart in these cases, however, showed no evidence of failure. The main cause of the hypertension is an increased blood volume, the output of the heart is increased due to this, with a correspondingly elevated systolic pressure and the diastolic pressure is raised purely on account of the increased blood volume. To accommodate such an increased blood volume, the vessels throughout the body dilate. The cerebral arterioles take part in this just as do the other vessels elsewhere.

The polycythaemia with its increased viscosity of the blood will further elevate the blood pressure. It will also tend to predispose to thrombosis.

The increase in blood volume together with the increased viscosity of the blood will slow the

venous circulation and thus impair the drainage of cerebro-spinal fluid especially that C.S.F. displaced by cerebral arteriolar dilatation. There is thus, an increase in the C.S.F. pressure, with stretching of the meninges and also cerebral arteriolar dilatation.

The obesity will increase the work of the already hypertrophied heart which perhaps, due to mild inefficiency will tend to raise the Co_2 tension of the blood, thus further producing cerebral arteriolar dilatation.

The altered personality may produce some psychological effect which may or may not predispose to headache in these cases.

2. Precipitating factors:

The precipitating factor in both cases was effort either vomiting or straining at stool or sudden rising from bed. This produced the typical throbbing frontal headache. This effort would increase the cardiac output, increase the systolic pressure, increase the force acting on the already dilated cerebral arterioles and due to the quickened heart-rate, increase the circulation rate of this large blood volume, resulting in sudden over-distension of the dilated cerebral arterioles. The polycythaemia with the increased viscosity would tend to slow the blood flow in the veins, thus preventing drainage of excess cerebro-spinal fluid displaced by this sudden arteriolar dilatation.

These two factors (1)The sudden over-distension of the arterioles and (2) the impaired cerebro-spinal fluid drainage would be sufficient to produce headache in a patient already in a disturbed psychological state. These mechanisms are explained more readily in the following diagrams.

Diagram B:

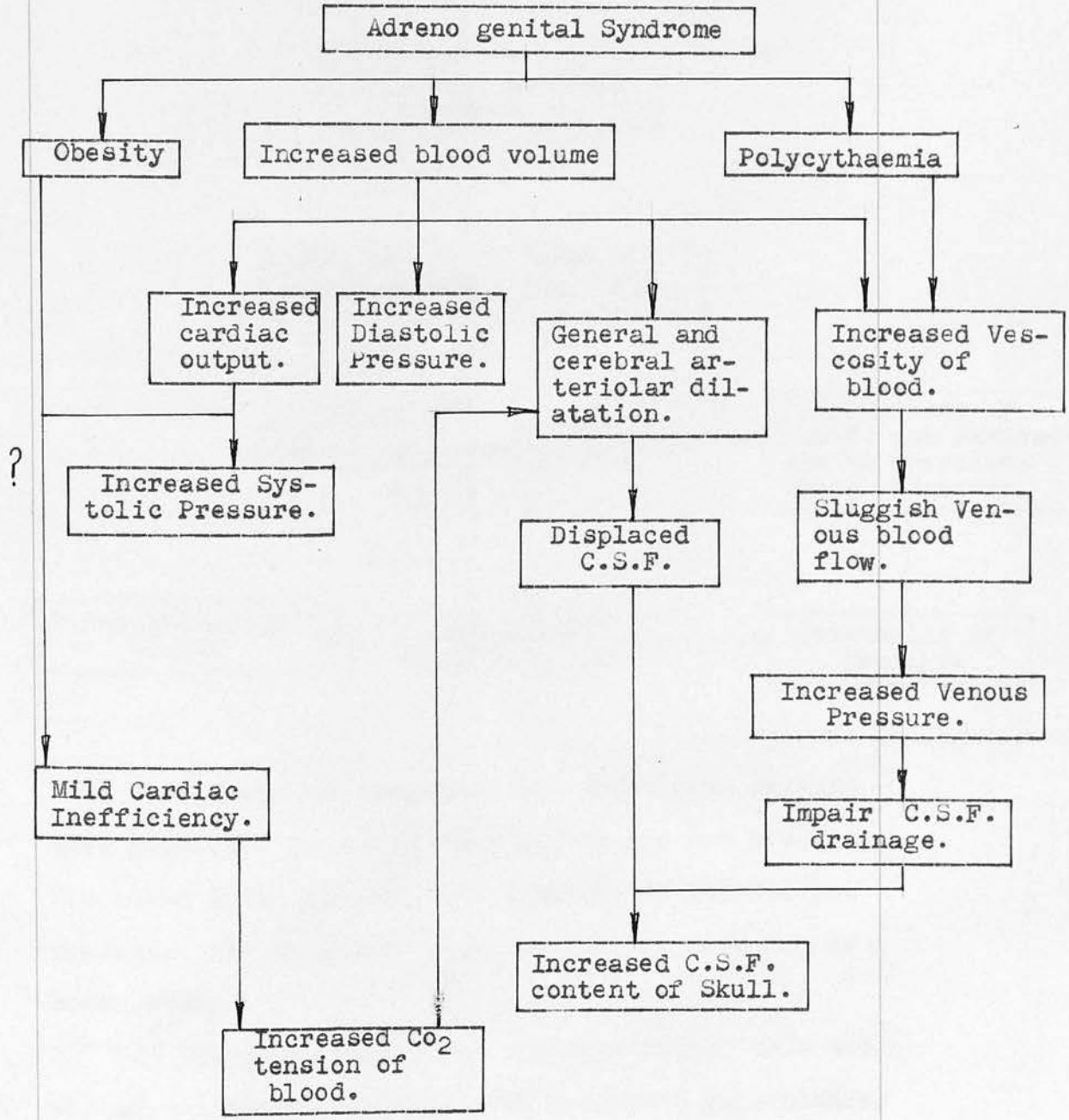
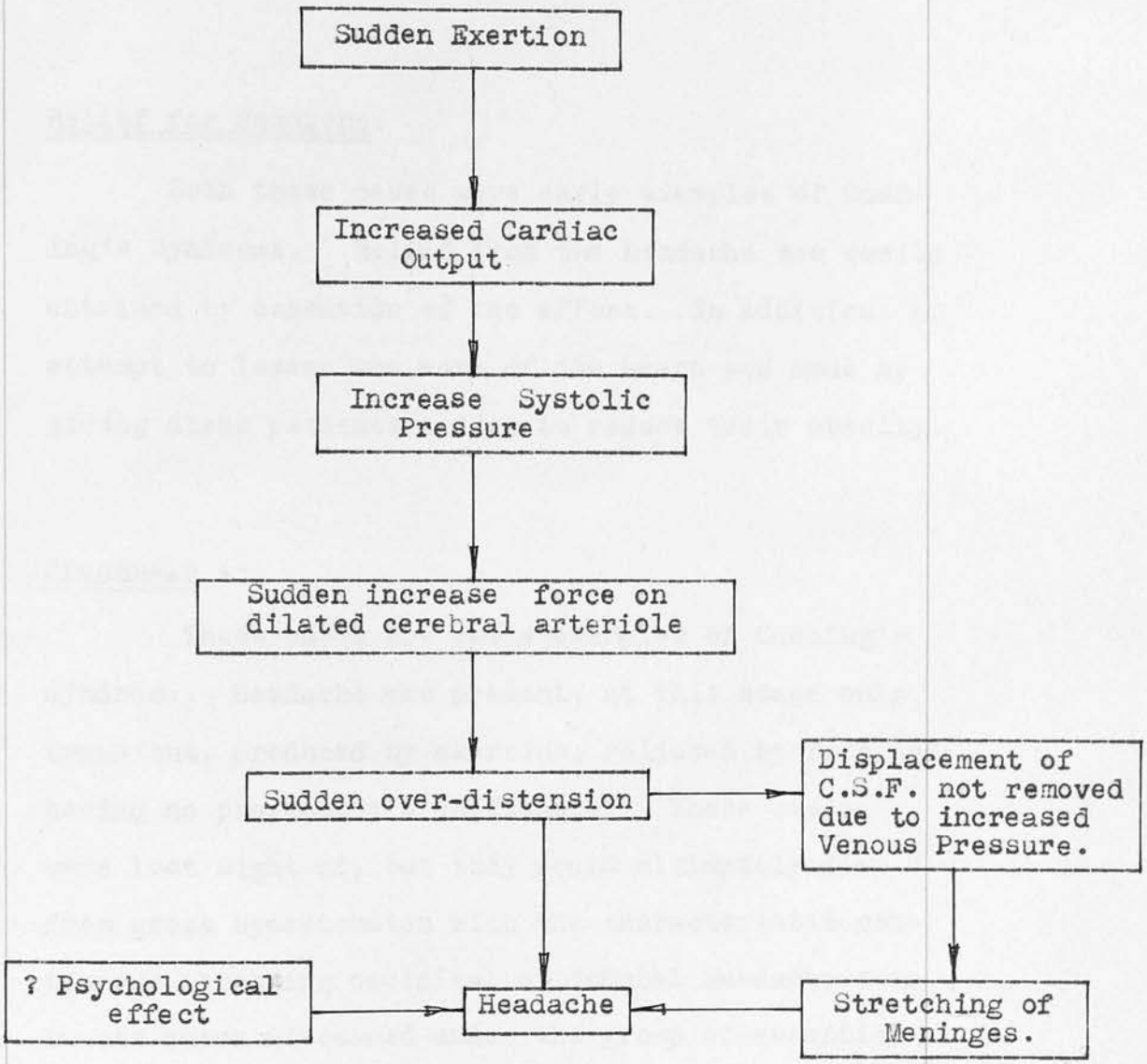


Diagram C:



In these two patients, two additional factors were present. In one patient glycosuria was present. The blood sugar was only 194 mgm% and no ketosis was present. The effect of this, therefore, need not be considered.

The other factor was/cholecystitis; this too /chronic was an incidental finding. The patient's gall-bladder

symptoms improved after operation and here again this factor does not enter into the discussion.

Relief for Headache:

Both these cases were early examples of Cushing's Syndrome. Relief from the headache was easily obtained by cessation of the effort. In addition, an attempt to lessen the work of the heart was made by giving these patients a diet to reduce their obesity.

Prognosis :-

These cases are early examples of Cushing's Syndrome. Headache was present, at this stage only transient, produced by exertion, relieved by rest and having no prognostic significance. These cases were lost sight of, but they would ultimately die from gross hypertension with the characteristic continuous throbbing occipital or frontal headache seen in the cases discussed under the group of essential diastolic hypertension.

8. Cases of Menopausal disturbance.

There are at least two pronounced changes in the hormone pattern which develop at the menopause, an under secretion of ostrin and an over-production of gonadotrophin. Zondek has shown that the gonadotrophic hormone not only continues to be formed after oophorectomy but is excreted in increased amounts. Albright has demonstrated that hypo-oestrinism is not the direct cause of the vaso-motor phenomena seen at the menopause. He further states, that œstrin therapy nevertheless, relieves the vaso-motor phenomena, and that oestrin acts by lessening the over-production of gonadotropic hormone.

In addition to this change in the hormonal pattern, the woman undergoes in some instances certain psychological or perhaps behaviour disorder.

Furthermore, it is a known fact that hypertension is encountered at the time of the menopause. This may be, because the hypertension was there already and is discovered on routine examination at this time. Another possibility is, that the change in the hormonal pattern, plus the associated psychological disturbance, results in excessive tone or hyperactivity of the sympathetic vaso-motor mechanism and results in hypertension. Unless hypertension exists previously, the production of a temporary high blood pressure at the menopause has, as far as the author knows, never been satisfactorily explained.

The cases of menopausal disturbance are analysed in Tables XXXIII & XXXIIIA (Appendix cases no. 41-48).

Analysis of Cases of Menopausal Disturbance.

Table XXXIII.

Sex	Famale	Female	Female
Age	48	46	45
Weight	9 st. 4 lbs.	12 st.	10 st.
Family History	Not relevant	Not relevant	Mother - Hypertensive.
Previous Illness	Not relevant.	Not relevant.	Not relevant.
Menstrual History	Stopped since 6 mths.	Irr. since 2 mths.	Stopped since 2 mths.
Presenting Symptoms	Headache flushing and sweating- 3 mths.	Heart-burn, headache, and giddiness - 3 mths.	Vague abd. pain. Giddiness and headache - 1½ years.
Headache	+	+	+
Duration	3 mths.	3 mths.	1½ mths.
Site	F.	F.	F.
Character	T.	T.	T.
Onset	Worse in morning.	In morning.	In morning.
Severity	M.	M.	M.
Duration of Attack	About ½ hour.	About 20 minutes.	½ hour.
Precip. factors	Rising from bed.	Sudden movement	Straining at stool.
Assoc. Symptoms	Flushing and Sweating	Flushing and Giddiness	Flushing, sweating.
Relief	Phenobarbitone and Stilboestrol.	Phenobarbitone, Stilboestrol.	Phenobarbitone, Stilboestrol.
B.P. Systolic / Diastolic	A 180 / 100 D 140 / 90	190 / 100 190 / 100	150 / 100 120 / 80
Heart	N.	N.	N.
Abdominal Examination	Negative.	Negative.	Negative.
Fundus Oculi	N.	N.	N.
Urine	N.	N.	N.
Treatment	Sedations and Stilboestrol.	Dietetic - 1200 calories Phenobarbitone, Stilboestrol and Mag. Trisilicate.	Dietetic Phenobarbitone. Stilboestrol.
Result	R.	R.	R.

Key:- + = Yes. F + Frontal. T = Throbbing. M = Moderate. N = Normal. R = Relief.
A_2 ++ = 2nd Aortic accentuated

Analysis of Cases of Menopausal Disturbance.

Table XXXIII[A].

Sex	Female	Female	Female	Female	Female
Age	47	41	46	46	48
Weight	10 st. 2 lbs.	9 st.12 lbs.	10 st.2 lbs	11 st.	9 st. 10 lbs.
Family History	Not relevant	Mother - Hypertensive	Not relevant	Mother - died Uterine cancer	Father - Cerebral Haem.
Previous Illness	Nil	Nil	Nil	Not relevant	Nil
MenstrualHistory	Irr. since 3 months	stopped since 3 mths.	Stopped since 2 mths.	Stopped since 1 mth.	Irr. since 6 mths.
Presenting Symptoms	Heading and Flushing	Nervousness, headache and giddiness.	Headache and Flushing	Flushing and Palpitation	Dyspnoea on exertion and headache.
Headache	+	+	+	-	+
Duration	2 mts.	2 mts.	2½ mts.	-	3 mts.
Site	F	F	F	-	F
Character	T	T	T	-	T
Onset	On rising	Worse in morning	During day	-	Worse in morning
Severity	M	M	M	-	M
Duration of Attack	About ½ hr.	½ hr.	½ hr.	-	About 1 hr.
Precip. Factors	Sudden movement of head	Sudden rising from bed	Stooping	-	Sudden movement of head
Associated Symptoms	Flushing	Flushing, Sweating	Flushing	-	Flushing
Relief	Phenobarbitone Stilboestrol	Phenobarbitone Stilboestrol	Phenobarbitone Stilboestrol	-	Phenobarbitone Stilboestrol
B.P. A D					
Systolic	160 140	160 140	150 130	160 140	160 160
Diastolic	105 90	110 90	100 85	100 90	110 110
Heart	N	N	N	N	A_2 ++
Abdominal Exam.	Negative	Negative	Negative	Negative	Negative
Fundus Oculi	N	N	N	N	I.
Urine	N	N	N	N	N
Treatment	Phenobarbitone Stilboestrol	Phenobarbitone Stilboestrol	Phenobarbitone Stilboestrol	Phenobarbitone Stilboestrol	Phenobarbitone Stilboestrol
Result	R	R	R	R	R

Summary of Menopausal Disturbance:

1. The total number of cases are eight.
2. The patients with headache - 7 cases out of 8 i.e. 87%.
3. Average age of patient with headache - 46 years.
4. Average weight of patient with headache - 10 st. 2 lbs.
5. Before Treatment Average B.P. $\frac{164}{103}$ mm Hg.
6. After Treatment " " $\frac{145}{92}$ mm Hg.
7. Headache:- Site - Frontal in 7 cases.
8. Character: Throbbing in all cases. It was always present on arising in the morning in 6 of the 7 cases. In remaining case it occurred during the day.
9. Precipitating Factors: It would appear that precipitating factors were viz:-
   (a) A sudden movement of the head in 2 cases and in one by sudden movement from posture to posture.
   (b) The patients suddenly rising immediately after awakening in 2 of the cases.
   (c) A stooping while doing housework in one case.
   (d) A straining at stool in one case.
10. Relief: Relief was obtained from headache at the same time as the flushing ceased and under treatment with:
   (a) Phenobarbitone.
   (b) Stilbœstrol.

In no case was there a previous history of any pre-eclamptic symptoms and in three cases there was a family history of hypertension. In no case, on examination, was the heart enlarged, and only in one case, was there Stage I. changes in the fundus. In no case was there any abnormality in urine.

Preliminary Conclusion :

1. The headache associated with Menopause in the above cases was situated and was of a similar character to that seen in Essential Hypertension.

2. The headache was associated with the vascular abnormalities, occurring during the menopause; for relief was obtained as soon as the vaso-motor instability improved with stilboestrol therapy.

3. In only two cases of the seven, was there any evidence of hypertension and this was only grade I. fundal changes, in one case and persistence of the blood pressure level in both cases after treatment. (Urinary and cardio-vascular system were normal).

4. There was no history of hypertension but these cases could not be followed up to ascertain whether they developed Hypertension in a few years time.

5. The facts that the blood pressure fell considerably viz. Systolic fell practically 20 mm Hg., and the Diastolic 11 mm Hg., would suggest that there was no permanent changes in the blood vessels during the menopause.

Discussion of these cases of Headache at the time of the Menopause:

The cases of menopausal disturbance can be divided into two groups :

A. Where there is a pre-existing hypertension, which remained after treatment of the menopausal symptoms - 2 cases (Nos. 42 and 48.)

B. Where the hypertension was transient and the blood pressure returned to normal after treatment of the menopausal disturbance 5 cases (Cases Nos. 41, 43, 44, 45 and 46).

In both groups, the character, site and duration of the attack of headache were similar. One can thus assume therefore, that the headache in this

series of patients was associated with the presence of the hypertension, whether permanent or transient, whether produced by whatsoever, is the cause of essential hypertension or whether resulting from the menopause.

In both groups of cases, one is dealing with a combination of (1) the results of the change in the hormonal pattern of the body;

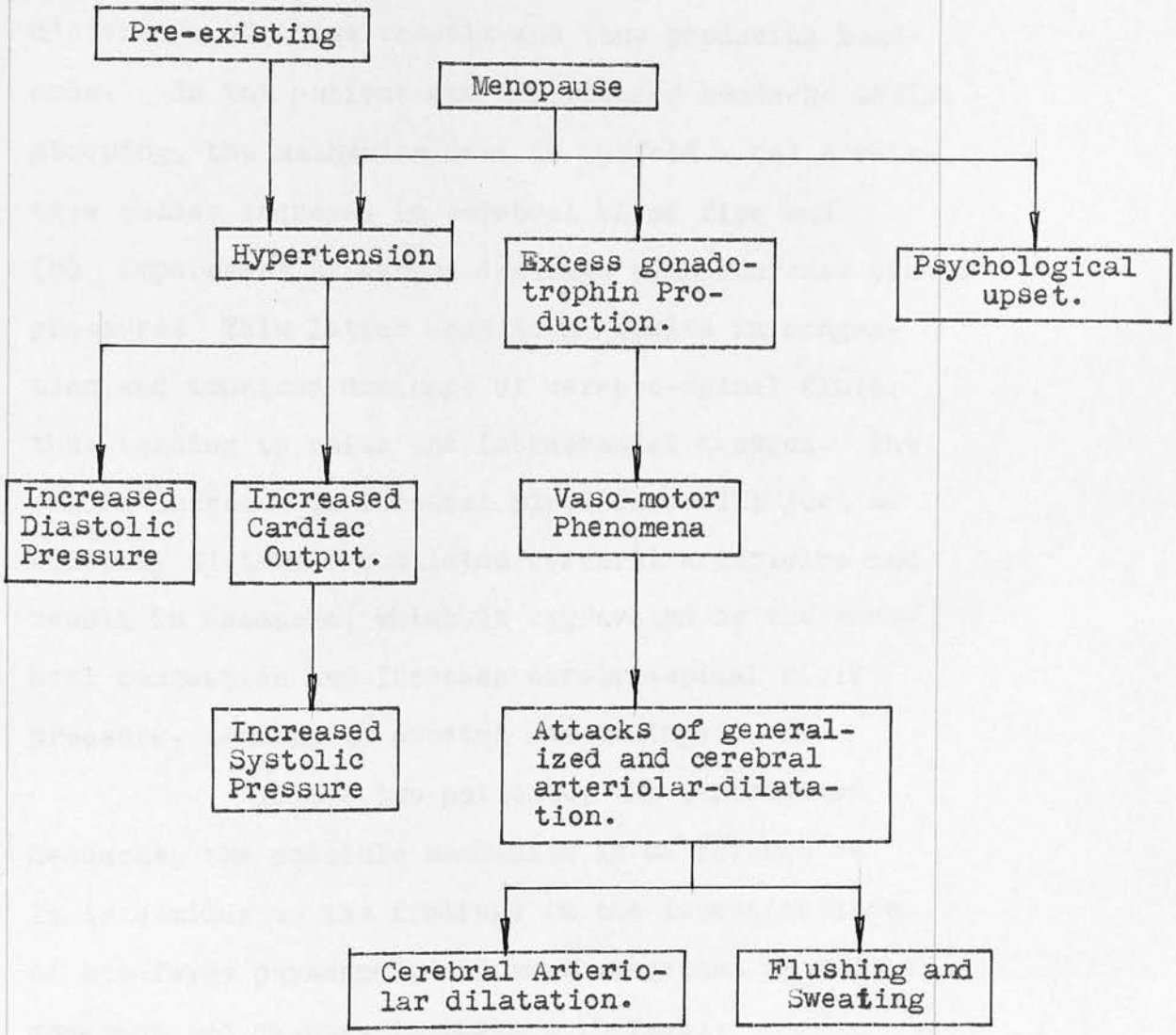
(2) The effects of hypertension, permanent or transient.

It is known that the flushing, sweating and other uncomfortable sensations experienced at the menopause are due to undue irritability of the smaller arterioles (Zondek and Albright) resulting from excess production of gonadotrophic hormone. In addition, hypertension is present.

There is an increased cardiac output and increase therefore, in the systolic level and an increase in the diastolic pressure due to hypertension.

The predisposing factors in these cases therefore, are the excessive production of gonadotrophic hormone resulting in sudden generalized, including cerebral arteriolar dilatation (experienced by the woman as hot flushes), together with an increased systolic and diastolic pressure. No case in this series, had cardiac or renal complications of hypertension, thus the permanent changes in the body resulting from hypertension play no part in the mechanism in these cases.

A combination of the many hormonal disturbances causes psychological effects which must be considered in headache production.



The Precipitating factors in all these cases was some exertion of some kind or another. In two patients, it was brought on by suddenly rising from bed, in two others by sudden movement of the head, in yet another, by sudden movement from one pos ture to another, in one woman by straining at stool and

in the final case by stooping. Exertion in these four cases, would result in a sudden increase in cardiac output, or sudden elevation of the systolic pressure and therefore, a sudden increase in force acting upon dilated cerebral arterioles with over-distension of these vessels and thus producing headache. In the patient who experienced headache whilst stooping, the mechanism here is twofold - (a) A relative sudden increase in cerebral blood flow and (b) impairment of Venous drainage with increase venous pressure. This latter condition results in congestion and impaired drainage of cerebro-spinal fluid; thus tending to raise the intracranial tension. The sudden increase in cerebral blood flow will just as suddenly distend the dilated cerebral arterioles and result in headache, which is aggravated by the cerebral congestion and increase cerebro-spinal fluid pressure, tending to stretch the meninges.

In the two patients, who experienced headache, the possible mechanism is as follows :- It is similar to the findings in the investigations of air-force personnel, who were subjected to sudden movement and changes in posture (Barach).

This results in a sudden redistribution of blood in the brain. More blood is suddenly shifted in the vessels to that part of the brain in the direction of the sudden movement eg. - a sudden turning of the head to the right causes a sudden redistribution of the blood in the brain so that the

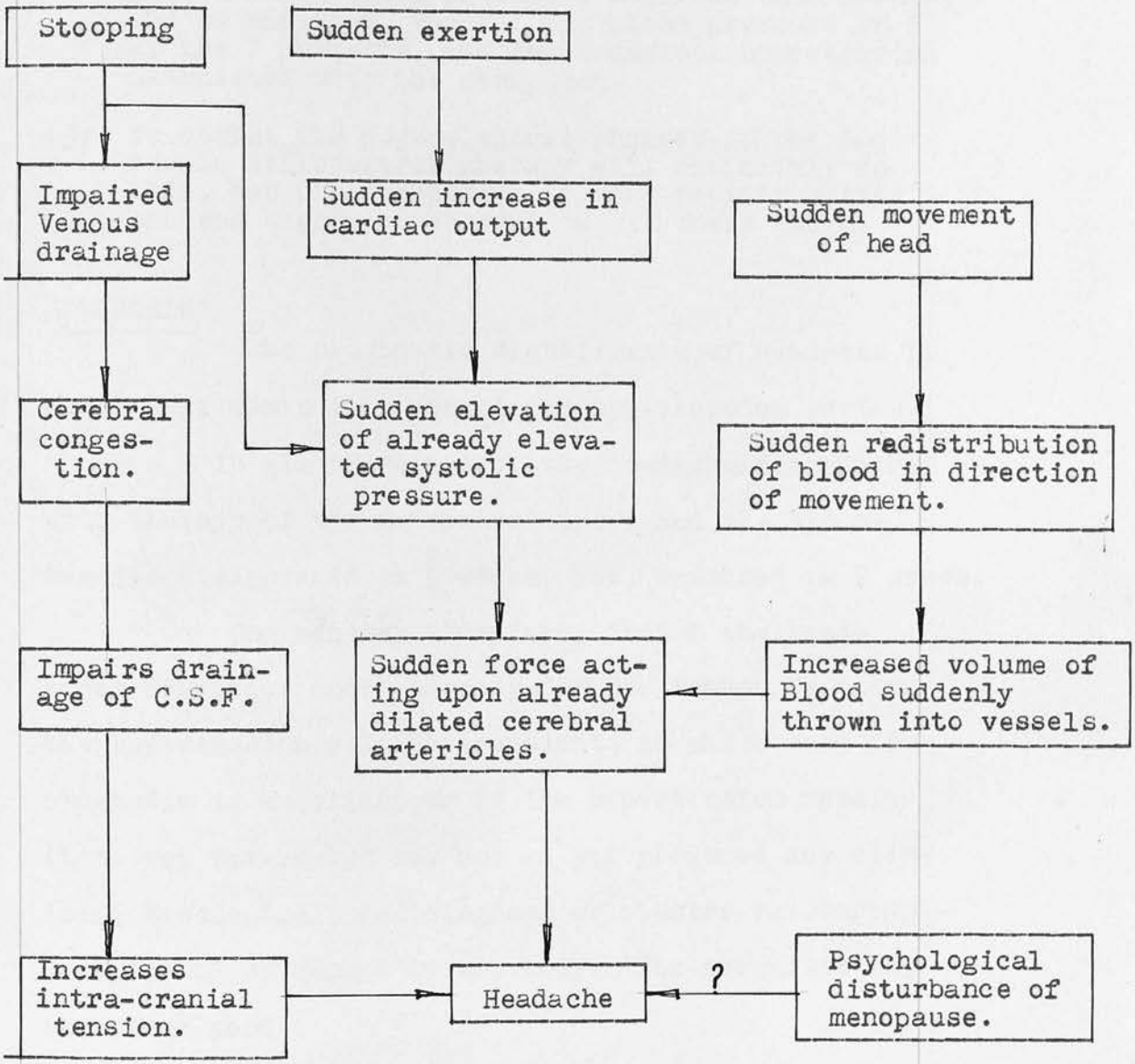
main volume of the blood is suddenly thrown into the vessels on the right of the brain. This sudden increase in blood volume in these vessels, results in sudden over-distension. which, no doubt, results in headache.

To summarize, therefore, the predisposing factors in the mechanism of headache associated with the menopause are cerebral arteriolar dilatation, increased blood pressure and associated psychological upset.

The precipitating factors are a sudden increase in the already elevated systolic pressure resulting from exertion or stooping, producing over-distension of these dilated arterioles, resulting from a sudden increase in blood volume, due to sudden movement.

These effects are seen in diagram (A)

Diagram A:



Relief of Headache:

This may be brought about in three ways:

(1) By means of gradual exertion, no sudden movements, and avoidance of straining at stool etc. This gave relief in all these cases.

(2) To render the vaso-motor mechanism less sensitive. This can be performed by reducing the quantity of circulating gonadotrophin (Zondek and Albright) The administration of oestrin in its many forms depresses the function of the anterior lobe of the pituitary gland and reduces the amount of gonadotrophic hormone produced, thus rendering the vaso-motor mechanism more stable. Stilboestrol

given in all these patients, achieved this result, and in addition, reduced the blood pressure in 5 of the 7 patients, who had transient hypertension associated with the menopause.

(3) To combat the psychological changes in the individuals stilboestrol therapy will ultimately do this, but phenobarbitone is more rapidly acting and was highly successful in all these cases.

Prognosis:

The prognostic significance of headache in menopausal women with associated hypertension varies.

In all these cases, the headaches disappeared with therapy of the menopausal upset and the hypertension disappeared in 5 cases, yet, remained in 2 cases.

One can say therefore, that if the headaches disappear under therapy for the menopause, then the hypertension will be transient, in which case the prognosis is excellent or if the hypertension remains, it is not severe and has not as yet produced any clinical, biochemical, radiological or electro-cardiographic evidence of damage in the body. The prognosis is, therefore good.

No cases were encountered by the writer in which the headaches did not disappear under therapy. One can assume however, that if the headaches did not improve under treatment of the menopause alone, then the hypertension would be severe, would have produced renal and cardiac complications and the prognosis would be grave.

B. SYSTOLIC HYPERTENSION.

The systolic element of the blood pressure is also elevated in some cases of diastolic hypertension, and hence requires ellucidation, so as to assess the importance of headache in such cases.

The significanee of headache in systolic hypertension is reviewed in the following cases, in view of the importance of contrasting it with diastolic hypertension, the significance of which has already been explained.

The cases in the series are 34,(one case of Alkalosis was excluded) and are explained as follows :

(1)	Aortic-regurgitation	-	10 cases.
(2)	Thyrotoxicosis	-	10 "
(3)	Atherosclerosis	-	10 "
(4)	Complete Heart-block	-	4 "

(Appendix: Cases 49-82

In the above series, 12 patients had headache, of which, 3 with aortic-regurgitation, 4 with thyrotoxicosis, 3 with atherosclerosis and one with complete heart-block.

The incidence of headache was slight in the above group, indeed so slight, and transient that a full investigation of the type of headache could not have been made in these cases, as it did not inconvenience the patients to an extent that required a full description of its significance. It was also

not a presenting symptom in many of these patients and some indeed, had to tax their memory to give details of its character.

A brief summary of the features of the headache obtained in such cases are shown in the following tables:-

Comparison of Features of Headache in Patients with Systolic Hypertension.

Table XXXIV.

	History of Headache	Site	Character	Onset	Duration of Attack	Precip factors	Associated Symptoms	Relief
1.Thyrotoxicosis. 4 out of 10	1-10 mths	Frontal 3 Behind eyes 1	Throbbing	Exertion 3 Reading 1	Few minutes	Exertion and excitement	Nil	Rest and treatment of Thyrotoxicosis.
2.Aortic regurgitation 3 out of 10.	3-6 mts.	Frontal	Throbbing	After dizziness 1 Exertion 1	Few minutes - 2 Not known - 1	Exertion	Nil	Rest.
3.Atheroma of Aorta 3 out of 10	Extremely variable history	Frontal	Throbbing	Prior to confusion 1 Movement 1 Exertion during day - 1	Few minutes	Exertion	Nil	Spontaneous 2. Claimed no releif in 1.
4.Complete Heart-block 1 out of 4	3 years	Frontal	Throbbing	During exertion only	Few minutes	Exertion	Nil	Rest.

Incidence of Headache : 12 out of 34 cases - 35%.

Incidence of Headache in Patients with Systolic Hypertension.

Table XXXV.

	Thyrotoxicosis	Aortic regurgitation	Atheroma of Aorta	Complete Heart Block
Incidence	40%	30%	30%	25%
Sex-incidence	All Females	All Males	1 M : 2 F.	Female
Average Age	35 yrs.	46 yrs.	77 yrs.	35 yrs.
Average weight	8 st. 12 lbs.	10 st. 12 lbs.	8 st. 9 lbs.	8 st. 5 lbs.
Av. B.P. on Admission	168/77	186/73	197/87	160/70
On Discharge	132/72	180/73	185/78	140/70
Site	Frontal	Frontal	Frontal	Frontal
Character	Throbbing	Throbbing	Throbbing	Throbbing
Precip. factors	Exertion	Exertion	Exertion	Exertion
Fundal Findings	Normal	Normal	I. (No significance)	Normal
Relief	Rest	Rest	Rest	Rest

The headache in these cases was a minor symptom, occurring during exertion, and the clinical impression, that the writer has gathered was, that the throbbing sensation caused by the sudden increase in pulse pressure on exertion, was interpreted by these patients as headache. It is possible, that cerebral vaso-dilatation played a part in the production of the headache, but the transient nature and infrequent occurrence of such a trivial symptom in these cases, did not necessitate full investigation and therapeutic measures.

The impression, therefore, would be, that headache in systolic hypertension has about as much significance, as the systolic hypertension itself. Clinically, systolic hypertension is of little importance, likewise, the transient headache of minor severity is not of much practical importance and therefore, can be disregarded.

The mechanism of production may be similar to that of diastolic hypertension, being cerebral arteriolar dilatation, resulting from anoxia.

FINAL CONCLUSIONS.

A. Diastolic Hypertension:

The mechanism of headache production in the many types of hypertension has been discussed following the analysis of clinical observations of 48 patients with diastolic hypertension.

The series are made up as follows :

(1) Essential hypertension	15 cases	- 75%	headache
(2) Chronic glomerulo-nephritis	10 "	- 90%	"
(3) Chronic Pyelonephritis	1 case		
(4) Amyloidosis	3 cases	- 66%	"
(5) Subarachnoid haemorrhage	5 "	- 100%	"
(6) Cerebral tumours	4 "	- 75%	"
(7) Cushing's Syndrome	2 "	- 100%	"
(8) Menopausal	8 "	- 87%	"

The incidence of headache, therefore, in diastolic hypertension is 41 of 48 patients. One patient in this series was discarded as it was thought that a concommitant haematemesis and resulting anaemia produce headache in this woman. Thus the corrected figures show that 83% suffered from headache.

In all these cases, the headache could be classified into two groups, frontal headache and occipital headache. Apart from the difference in location of the headache, the character of both types was identical. This throbbing headache, be it frontal or occipital, in every case was brought on, or made worse by rising in the morning. The headache

in all cases is dependent upon a vascular mechanism and a vascular mechanism only. Dilatation of the cerebral arterioles is the main determining factor in the majority of these cases for the production of headache.

Investigations on these patients with headache, and from observations on other patients during sleep, have shown that acidaemia is the chief factor, which produces the vaso-dilatation, resulting in headache in patients with essential hypertension, chronic glomerulo-nephritis, chronic pyelo-nephritis, amyloidosis, Cushing's Syndrome and Menopause. Although, in this latter condition, excess of gonadotrophine play a large part in producing vascular dilatation. In Cushing's Syndrome increased blood volume is an important factor in producing vaso-dilatation. In some patients suffering from cerebral tumours and subarachnoid haemorrhage, traction on cerebral vessels, remote from the site of the lesion plays a part in the dilatation. In addition, of course one must not overlook the irritant effect of the extravasated blood on the meninges, producing the typical meningeal headache in subarachnoid haemorrhage.

Thus dilatation of the cerebral arterioles due to acidaemia is the main determining factor in the majority of these cases. The precipitating factor is the sudden increase in cardiac output consequent on rising, which produces over-distension of these dilated cerebral arterioles and results in pain.

Our observations have determined, that the acidaemia in the two types of headache is different. In the frontal headache, the incidence of headache, its severity and the relationship between the many factors present in these cases at the time of examination, stress the point that cardiac insufficiency with its increased Co_2 tension, is the main factor in production of acidaemia. The acidaemia is increased by the accumulation of acid radicles due to renal impairment and both these factors are exaggerated by mild physiological acidaemia of sleep.

In the occipital headache, the clinical observations have shown that renal failure is the main theme running through all these cases, the incidence, severity and relationship with other factors are all dependent on renal insufficiency. The acidaemia in these cases is therefore, due to the acid radicles, which the kidney cannot eliminate. Cardiac decompensation and sleep with their resulting acidaemia play only minor rôles.

Furthermore, clinical investigation has shown that the duration of the headache is dependent upon renal function. The longer the attack of headache lasts, the more severe is the kidney damaged.

An idea as to the prognosis can also be obtained by observing the severity and duration of the headache and its site. Frontal headache of great severity and of long duration, indicates a bad prognosis

and most certainly cardiovascular complications whether renal complications be present or not. Severe persistent occipital headache has a grave prognosis and indicates gross renal failure.

The explanation as to the different sites of the headache is difficult, if not impossible. Psychological factors, other minor lesions such as fibrositis or cervical osteoarthritis, eyestrain and even a different response of the cerebral arterioles to the type of acidaemia must be considered.

B. Systolic hypertension:

The results of the analysis of 34 patients with elevation of the systolic pressure only, showed that only a few complained of headache. The headache was a minor symptom only and was never a presenting symptom. As far as can be told, the headache has no significance. Its mechanism may be similar to the headache production in diastolic hypertension, namely due to cerebral arteriolar dilatation. In the patients with systolic hypertension, it is suggested that the anoxaemia caused by their exertion produced the vaso-dilatation, although proof of this could not be obtained due to the transient character and infrequency of attacks of such a minor complaint.

The initial observation by the author, of the relief of headache, by a tight folded

handkerchief around the head just above the ears, has led him far and wide into the realms of physiology, pathology and biochemistry. Probably these patients with their handkerchiefs, obtained relief simply by the counter-irritation of "circumcranial compression", although, one could postulate lessening of the arterial supply to the site, where pain is felt or even impairment of lymph and venous drainage of the scalp and via emissary veins of the cerebrum, with the prevention or lessening therefore, of over-distension of cerebral vessels as possible explanations.

This "circumcranial compression" will most certainly impress the patient that he is receiving direct treatment to the seat of his trouble rather more than will investigating his blood urea, eye-grounds or cardiac function ! For the clinician, however, the latter investigations are of much more value.

SUMMARY.

1. The literature on the causes of headache and the aetiology, classification and pathological effects of hypertension have been briefly reviewed.

2. Clinical observations have been made on eighty-two patients suffering from many types of diastolic hypertension (48 cases) and systolic hypertension (34 cases).

3. The results of these observations have been analysed and the incidence and severity of the headache have been correlated with the pathological changes and clinical findings at the time of examination.

4. The degree of acidaemia of patients with cardiac failure during waking hours and at the time of wakening have been compared with a control series.

5. An attempt has been made to explain the mechanism of headache in these cases based upon clinical examination together with the known experimental findings of other workers.

6. A purely vascular mechanism for the headache is suggested in these cases.

7. The basis of this headache is cerebral vasodilatation. This may result from the many causes of acidaemia, from traction on the vessel, from excess gonadtrophines and from direct irritation. The precipitating factor is a sudden over-distension of these dilated vessels caused by a sudden movement of the head, sudden exertion, or sudden increase in cardiac output.

8. The significance, prognosis and mode of relief in these cases are discussed.

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APPENDIX.

Case History.

Essential Hypertension:

Case 1.

Male, aged 51, railway-telephone worker, married, weight 10 sts. 12½ lbs.

Complaint: (1) Tiredness and fatigue, 18 months; (2) History of Influenza 6 months ago; (3) Tight feeling in chest for the last 2 months occurring during the day, disappearing by rest; (4) No headache.

Previous Illness: Pneumonia 5 years ago.

Habit: Smoker of cigarettes.

Family History: Father died at 66 years of gastric cancer. Mother aged 74, alive and diabetic. Wife alive and well. Daughter aged 21, alive and well.

General Examination: Well-built man of average intelligence, does not appear to be in great distress, but nervous on examination.

Cardio-Vascular System : Pulse rate 90 per minute, regular in time and force, arterial wall not palpable.

Blood Pressure on admission: $\frac{260}{160}$ mm Hg.
No visible pulsation on neck.

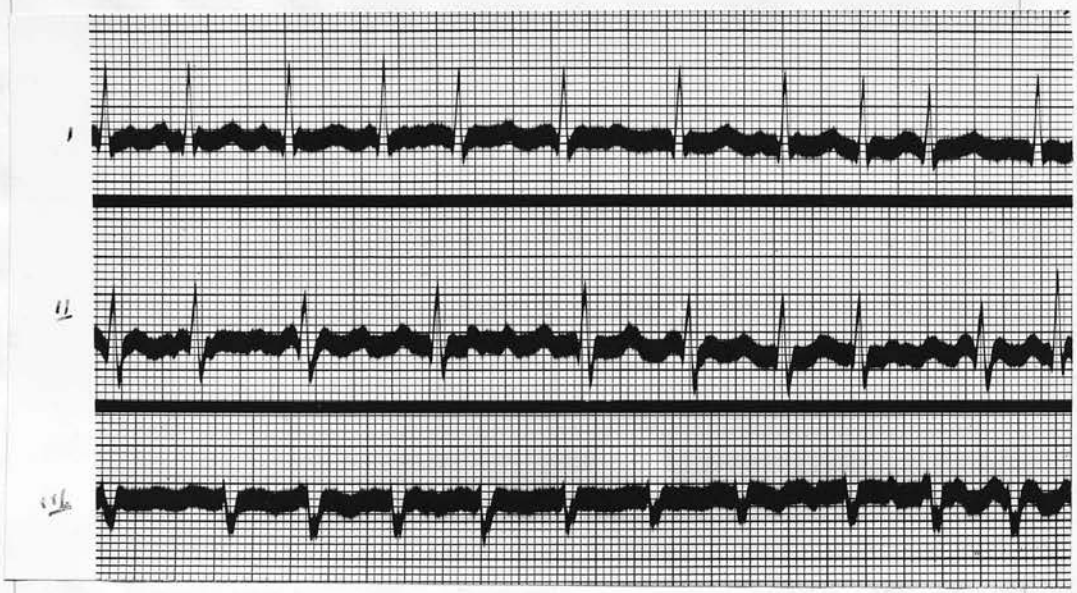
Heart:
Apex beat : 5th left intercostal space, one inch outside to midclavicular line.
Ausculation : Accentuated first mitral, soft systolic murmur at the aortic area with reduplication of the 2nd sound.

Respiratory System: Nil to note.

Liver and Spleen - not palpable.

Investigations:
(1) X-Ray of the heart: Enlarged left ventricle, Prominent aortic arch and calcification of aorta.
(2) E.C.G.: Left axis deviation.
(3) Urine vol. 1550 c.c. in 24 hours. Acidic, sp.gr. 1016, Traces of sugar, Albumin Nil.

CASE N° 2



Urea Range: Concentrated urine sp. gr. 1015 - urea 3·2 gm%.
Dilute Urine sp. gr. 1010 urea 0·2 gm%.

Blood Urea Nitrogen: 15 mgm%.

W.R. : Negative.

Fasting Blood Sugar: 186 mgm%

Sodium Amytal Test: Blood pressure fell from $\frac{220}{110}$ to $\frac{110}{80}$. Fundi: Grade I.

Diagnosis: Essential Hypertension and Diabetis Mellitus.

Treatment: Bed, rest, Phenobarbitone gr. ½ t.i.d.orally, 2400 calories diet with 28 units of zinc protamine Insulin in a day.

Result: Became sugar free, and relief of symptoms. Pulse rate 80 per minute and regular on discharge. B.P. $\frac{160}{110}$ mm Hg. on discharge.

Case 2.

Male, aged 66, Fire-place builder, married. weight 10 sts. 6 lbs.

Complaint: (1) Complains of pain along the back of the left thigh and left leg for 2 weeks; (2) Benumbed feeling in both legs for the last 5 years; (3) Moderate Throbbing headache in frontof the head on rising in the morning, lasting for about ½ hour, duration 6 months; (4) Sleeplessness at night and wakes up with breathlessness since a few weeks.

Previous Illness: History of Sciatica in left leg 20 years ago.

Habit: Smoker of Cigarettes.

Family History: Not available.

General Examination: Pale, anxious expression and confused memory.

Cardiovascular System : Pulse rate, 90 per minute, irregular in time and force, vessel wall palpable.

Blood Pressure on admission $\frac{240}{130}$.

Apex Beat: 6th left intercostal space in anterior axillary line.

Auscultation: Second aortic accentuated.

Lungs : Crepitation in both bases.

Investigations: X-Ray of heart; Enlarged left ventricle. E.C.G. Left axis deviation with auricular fibrillation; Fundi Grade III.

Urine Volume in 24 hours 1500 c.c. Acid Reaction, sp.gr. 1018; Albumin Nil; Casts Nil.

Blood Urea Nitrogen: 20 mgm%.

X-Ray of Legs: Calcification of both Posterior Tibial arteries.

X-Ray of Lumbar-Spine: Osteo-Arthritic changes.

Treatment: Bed, rest in "head-up" position whenever required. Phenobarbitone gr. ½ T.i.d. by mouth. Aminophylline 0·2 gm. T.i.d. and also at night.

Result: (1) Improved and discharged symptom free three weeks after admission; (2) Blood pressure $\frac{240}{130}$ unaffected in this case. Headache relieved. Pulse Rate 90 per minute and irregular on discharge.

Case 3.

Male, aged 71, Stone-Mason, Married, weight weight 8 sts. 9½ lbs.

Complaint: (1) Cough with spitting for the last 5 years; (2) Progressive breathlessness on exertion since 5 years, aggravated during the last 6 months; (3) No Headache.

Previous Illness:- "Influenza" followed by Pleurisy in 1918.

Habit: Non-smoker.

Family History:- Father at the age of 77 died of accident.
Mother, died at 80 of Old Age.
Wife aged 70, alive and well.
Children, seven, all alive and well.

Physical Examination:- A red-faced man, slightly orthopnonic.

Pulse rate:- 80 per minute, regular in time and force. Vessel wall thickened and tor

Blood Pressure on admission $\frac{220}{100}$.

Inspection:-

Apex-beat 5th left inter space outside midclavicular line.

Auscultation:- Aortic and Pulmonary second sound accentuated.

Investigations:-
(1) X-Ray of Chest:- Silicolis of both lungs,; enlarged left ventricle; Calcification of Aorta; (2) E.C.G. Left Axis deviation; (3) Repeated sputum examination gave negative result for Tubercle bacilli; (4) Urine Volume 1600 c.c. Acid Reaction; sp.gr. 1012; No albumin and no Casts; (5) Fundi; Grade I.

Diagnosis: Essential Hypertension and Silicosis of Lungs.

Treatment:- Bed rest, Phenobarbitone gr. $\frac{1}{2}$ T.i.d. orally; Inhalation of Penicillin, and Breathing exercises.

Result:- Slight improvement. B.P. fell to $\frac{190}{100}$ on discharge; Pulse rate 80 per minute, and regular

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Case 4.

Male, 53, Fisherman, Married. The patient admitted in a state of unconsciousness with Cheyne Stokes respiration. The history obtained from the patient's wife was:

Severe frontal headache for the last 6 months, worse on rising in the morning. It was said to be throbbing in character, lasting for about $1\frac{1}{2}$ hours. No relief was obtained from the drugs.

Family History:- Wife aged 45, alive and well; children two, both alive and well.

Habit:- Heavy smoker and drinker.

On Examination:- The pupils were small but reacted to light. Left-sided hemiplegia, Temperature normal.

Pulse Rate:- 70 per minute, regular in time and force; Vessel wall not palpable.

Blood Pressure:- $\frac{240}{120}$.

Apex -beat:- 6th interspace one inch outside the mid-clavicular line.

Auscultation:- First sound booming at the mitral area.

Respiratory System:- Bilateral basal crepitation.

Investigations:- Catheterized specimens: Vol. 2400 c.c., Acidic, sp.gr. 1010, Albumin present; Hyaline casts present; Fundi: Grade IV.

Lumbar Puncture:- Pressure 400 mm H_2O; C.S.F.: Blood-stained.

Diagnosis: Cerebral Haemorrhage.

Result:- Died of Cerebral Haemorrhage. No Post-mortem was done.

Case 5.

Male aged 61, Undertaker, Married, weight 11 sts. 5 lbs.

Complaint:- (1) Breathlessness on exertion, one year. (2) Bleeding from the nose off and on for 8 years; (3) Throbbing occipital headache on rising in the morning lasting about an hour, duration one year. Moderate in severity and attacks relieved by Epistaxis.

Previous Illness:- Dysentery 1914, Erysipelas 10 years ago.

Habit:- Heavy drinker and smoker.

Family History:- Wife aged 54 alive and well; Father 43, died, cause not known. Mother died at 50 from Pneumonia.

On Examination:- Highly coloured, over-weight, man of average intelligence.

Cardio-Vascular System:-
Pulse Rate: 60 per minute, regular and vessel wall palpable.

Blood Pressure:- $\frac{220}{120}$ mm Hg on admission.

Apex beat:- 5th left interspace, one inch outside the Mid-clavicular line.

Auscultation:- Booming first sound at the mitral area. Systolic murmur at the aortic area and 2nd aortic sound accentuated.

Respiratory System:- Few Bilateral Basal Crepitations.

Investigations:- (1) X-Ray of heart:- Enlarged left ventricle with unfolding aorta;
(2) E.C.G. Left Axis Deviation;
(3) Urine:- Volume 2800 c.c. Reaction acid; sp.gr. 1018; Trace of Albumin; Hyaline Casts present; Fundi: Grade III.

Blood Urea Nitrogen:- 30 mgm%; Haemoglobin estimation: 105%.

Diagnosis:- Essential Hypertension.

Treatment:- Bed Rest in "head-up" position when required; Phenobarbitone gr. ½ T.i.d.
Venesection: ½ pint of blood removed.

Result:- Headache relieved by Venesection.
Pulse Rate 60 per minute - regular;
B.P. $\frac{180}{110}$ on Discharge.

Case 6.

Male, 38, Motor-driver, Married, weight 16 sts.

Complaint:- Increase in weight and Tiredness since 3 yrs.
Trouble with eyes - for 3 yrs.
Shortness of Breath- for 1 yr.
Throbbing frontal headache on rising in the morning lasting an hour moderate in severity, duration - 6 months.

Previous Illness : Nil.

Habit:- Smoker.

Family History:- Nil to note.

General Examination:- Heavily built, red-faced man, cheerful disposition; height 6 feet, and well proportioned; catarract in left eye.

Cardio-Vascular System: Pulse rate: 70 per minute, regular and vessel wall not palpable.

Blood Pressure: $\frac{160}{100}$ mm Hg. on Admission;
Apex beat: Not palpable.

Auscultation:- First sound heard beat in 5th left interspace outside the mid-clavicular line. Both sounds normal but splitting of the 1st sound at the mitral area after exercise.

Lungs:- Nil to note.

Investigations:-
X-Ray of heart:- Left Ventricular enlargement.
E.C.G. : Normal; Blood urea N_2: 18 mgm%.
Urine:- Volume 2000 c.c. in 24 hours, acid in reaction, sp.gr. 1020, no albumin and no casts.

Urea Range:- Con. specimen: sp.gr. 1020, urea 4 gm%.
Dilute specimen: sp.gr. 1010, urea 0·8 gm%.
i.e. (within normal limit).

Fundi:- Grade I. in right eye, and catarract in left eye.

Diagnosis:- Essential Hypertension.

Treatment:-(1) Bed rest and "head-up" position when required; (2) Phenobarbitone gr. ½ T.i.d. orally; (3) Diet:- 1500 Calories, weight reduced to 15 st. 10 lbs. in 14 days.

Result:-(1) Weight very slightly reduced; (2) B.P. $\frac{150}{100}$ mm Hg on discharge; (3) Headache relieved; (4) Pulse Rate 70 and regular.

Case 7 :-
Male, 69, Merchant, Married, wt. 14 st. 10 lbs.

Complaint:- (1) Passing of bright red blood in stool for a day; (2) Attacks of giddiness and throbbing occipital headache of moderate severity coming on frequently, at any time for the last year, aggravated on wakening in the morning and straining at stool, and lasting several hours. Relief obtained by Aspirin; (3) Breathlessness on exertion for the last year.

Previous Illness:- Nil to note.

Habit:- Smoker and Drinker.

Family History:- Wife aged 62, two daughters and two sons, all alive and well.

General examination:- Heavy built, fairly intelligent.

Cardiovascular System:- Pulse rate: 90 per minute, regular and vessel wall palpable.

Blood Pressure:- $\frac{210}{120}$ mm Hg on admission.

Apex Beat:- 5th left intercostal space, one inch outside the M.C. Line.

Auscultation:- Heart sounds faint due to Emphysema.

Respiratory System:- Rhonchi in both lungs.

Investigations:- (1) Rectal examination revealed no abnormality; (2) Barium Enema: Nil to note; (3) W.R.: Negative; (4) Blood Urea Nitrogen: 24 mgm%.; (5) Fundal Exm.: Grade I.; (6) Urine: Volume 1900 c.c.,Acidic. sp.gr. 1016; No albumin, No casts.

Diagnosis:- Essential Hypertension.

Treatment:- (1) Bed rest and "head-up" position when needed; (2) Phenobarbitone gr. $\frac{1}{2}$ T.i.d. orally.

Result:- No headache while in Hospital (3 weeks);
B.P. $\frac{210}{120}$ mm. on Discharge.
Pulse rate: 65 per minute and regular.

Case 8:-
Female, 76, Housewife, Married, Wt. 9 st.

Complaint:- Collapsed while at work, the day before admission.

History:- (1) Known to have high blood pressure for the last 5 years; (2) Breathlessness on exertion and attacks of giddiness always followed by slight throbbing occipital headache on rising from bed in the morning, but not inconvenienc-ing the patient; duration one year. No other complaint.

Previous Illness:- History of Peptic Ulcer 11 years ago, and medically treated.

Family History:- Husband at 80 died of cancer of liver. Three children alive and well; Mother aged 64, died of cerebral haemorrhage.

General Examination:- Thin, pale old-looking woman.

Cardiovascular System:- Pulse rate: 80 per minute, regular and vessel wall thickened.
Blood Pressure:- $\frac{160}{110}$ mm Hg on admission.

Heart:- Apex-beat:- 5th left Interspace, within Mid-clavicular line. Auscultation: Aortic second sound accentuated.

Respiratory System:- Nil to note.

Investigations:- (1) X-Ray of heart:- Calcification of aorta; (2) E.C.G.: Normal; (3) Blood Urea Nitrogen:- 16 mgm %.; (4) Fundi: Grade I.; (5) Urine: Volume 2100, Acid in reaction; sp.gr. 1020; (6) Sodium Amytal Test: B.P. fell to $\frac{150}{90}$ (7) Urea Range: Con. specimen 1030 - 4 gm%. Dilute Specimen 1006 - 0·4 gm%.

Treatment:- (1) Bed rest for 4 weeks and "head-up" position when required; (2) Phenobarbitone gr. ½ T.i.d. by mouth.

Diagnosis:- Essential Hypertension.

Result:- (1) Headache relieved; B.P. $\frac{160}{100}$ mm Hg on Discharge; (3) Pulse Rate 80 per minute, and regular on discharge.

Case 9 :-
Female, aged 72, Housewife.

Complaint:- (1) Admitted with left-sided Hemiplegia due to cerebral thrombosis, gradually developed in 2 days; (2) Complained of throbbing occipital headache during the day and worse in the morning, associated with attacks of giddiness. The attack of moderate severity lasting several hours, duration 2 years, and claimed no relief; (3) Increasing breathlessness on exertion for the last 2 years; (4) Frequency of micturation at night since 2 years; (5) Mental confusion for 2 years.

Family History:- Not available.

Habit:- Nil.

On examination:- Pale. elderly woman with left-sided haemiplegia. Asymmetry of face was noticeable.

Cardiovascular System:- (1) Pulse rate: 100 per minute, regular and vessel wall palpable.
Blood Pressure:- $\frac{200}{110}$ mm Hg on admission.

Heart:- Apex beat: 5th left interspace just outside mid-clavicular line. Auscultation: 2nd Aortic sound accentuated.

C.N. System:- Pupils small and reacted to light and accommodation. Reflexes exaggerated in paralysed limbs (left arm and left leg). Left planter response:- Dorsiflexion; no sensory disturbance.

Investigations:- (1) Urine: Volume 3000 c.c. reaction acid, sp. gr. 1010, albumin +, Hyaline Casts +.; (2) Blood Urea N_2: 80 mgm%.; (3) Fundal Exm.: Grade III.

Diagnosis:- Left-sided hemiplegia due to cerebral thrombosis.

Treatment:- Bed rest, Penicillin, and Symptomatic.

Result:- Died of Hypostatic Pneumonia.

Case 10 :-
Female, 56, Housewife, Unmarried, wt. 10 st.4 lbs.

Complaint:- (1) Attacks of palpitation and Dyspnoea on exertion for the last 5 years;
(2) Breathlessness on exertion for 5 years.
(3) Breathlessness at night since 4 weeks;
(4) Throbbing frontal headache of moderate severity on rising in the morning, lasting about ½ hour. Duration 10 years; (5) Frequency of micturation at night for one year.

Previous Illness:- Cervical ribs rmoved 3 years ago.

Habit:- Nil.

Family History:- Brother died a year ago of High Blood Pressure.

On Examination:- Over-weight fat woman.

Cardiovascular System:- Pulse rate: 90 per minute, regular and vessel wall not palpable.
Blood Pressure: $\frac{180}{100}$ mm Hg on admission.

Heart:- Apex beat: 5th left interspace within Mid-clavicular line. Auscultation: Heart sounds normal.

Lungs:- Bi-lateral basal crepitation.

Investigations:- (1) X-Ray of heart: Left ventricular enlargement; (2) E.C.G.: Left Axis deviation; (3) Blood Urea Nitrogen: 25 mgm%.; (4) Fundal examination: Grade I.; (5) Urine: Volume 1500 c.c. Acidic sp. gr. 1016. Albumin trace and occasional casts present.

Diagnosis:- Essential Hypertension.

Treatment:-(1) Bed rest for 4 weeks and "head-up" position, when required; (2) Phenobarbitone gr. i T.i.d. orally, later reduced to gr.½ B.i.d.; (3) Aminophylline 0·2 Gm. T.i.d. and also at night.

Result:- Headache relieved. B.P. $\frac{180}{100}$ mm Hg on discharge. Pulse Rate 70 per minute and regular.

Case 11:-
Male, aged 53, Gardner, married, wt. 8 st. 5 lbs.

Complaint:- The patient admitted with haemetemesis from peptic ulcer, which was known to have had for the last seven years, and for which he was medically treated in Hospital.

History of Present Illness:- (1) Vomited blood 2 months ago; (2) Crushing pain over the precordium and below the ribs for the last two months, which was relieved by food; (3) No headache.

Previous Illness:- Peptic ulcer 7 years ago.

Habit:- Smoker 20 cigarettes a day, and drinker of beer only.

Family History:- Father died at 77; Mother died at 90; five brothers all alive and well; Three sisters died, one from coronary thrombosis, one died of gastric carcinoma, and one died of chronic bronchitis and catarrh.

On Examination:- A thin man, with anxious look.

Cardiovascular System:- Pulse Rate: 70 per min., regular, vessel wall palpable.
Blood Pressure: $\frac{195}{110}$ mm Hg on admission, but after recovery from haemetemesis B.P. rose to $\frac{200}{110}$ mm Hg.

Heart:- Apex-beat: 5th left interspace, within Mid-clavicular line. Auscultation: Mitral 1st and 2nd aortic sound accentuated.

Lungs:- Nil to note.

Investigations:-(1) X-Ray of heart: Enlargement of left ventricle, unfolding aorta; (2) E.C.G.: Left Axis deviation; (3) Urine: Volume 1600 c.c. in 24 hours, acidic, sp.gr. 1016. Albumin nil; (4) Urea Range:-
Con. specimen:- sp.gr. 1020 - 3 Gm%.
Dilute specimen :- sp.gr. 1006 - 0·7 Gm%.; (5) Fundal : Grade I.; (6) Blood Urea N_2 15 mgm%.; (7) Barium Meal revealed Duodenal Ulcer; (8) Fractional Test Meal: Hyperchlorhydria present.

Diagnosis:- Haemetemesis and Essential Hypertension.

CASE Nº 12

Treatment:- (1) Rest; (2) Morphia gr. ¼ S.c.l. on admission; (3) Fluids administered orally. Later treated for peptic ulcer. Mag. Trisilicate T.i.d. ulcer diet. Phenobarbitone gr. ½ T.i.d. for 2 weeks.

Result:- No blood transfusion needed in this case. (1) Haemetemesis stopped; (2) B.P. 200/110 on Discharge; (3) Pulse rate 70 per minute and regular.

Remarks:- The haematemesis resulted from duodenal ulcer, but aggravated by hypertension.

Case 12:- Male, 67, Retired School Master, Married, wt. 12st. 13 lbs. Complaint:- (1) Breathlessness on exertion, increasing rapidly for the last 3 weeks; (2) Getting up at night three times for urination for 3 months; (3) Moderate throbbing occipital headache rising from bed in the morning, lasting for an hour. Duration 13 years; (4) Breathless on exertion for 10 years; (5) Dizziness for 3 years.

Previous Illness:- Malaria 1939.
Habit:- Pipe smoker and drinker.
Family History:- Uneventful.

On examination:- Heavy built individual with average intelligence.

Cardiovascular System:- Pulse rate: 86 per min, regular and vessel wall not palpable. B.P. 180/100 on Admission. Heart: apex beat: In 6th left intercostal space one inch outside the mid-clavicular line. Ausculatation: Systolic murmur at the mitral area, Systolic murmur at the aortic area and 2nd aortic sound accentuated.
Lungs:- Bilateral basal crepitation.

Investigations:- (1) X-Ray of heart: Enlarged left Ventricle; (2) E.C.G.: Left Axis deviation; (3) W.R.: Negative; (4) Blood Urea Nitrogen:- 15 mgm%. (5) Fundal changes: Grade II.; (6) Urine: Total volume 1600 c.c. Acidic, sp. gr. 1013, albumin nil.

Diagnosis:- Essential Hypertension.
Treatment:- (1) Bed rest, for 4 weeks; (2) Phenobarbitone gr. ½ T.i.d. orally.

Result:- Headache relieved. B.P. 180/100 mm Hg. on discharge. Pulse Rate:- 70 per min. and regular.

Case 13:- Male, 48, Motor Driver, Married, wt. 12 st. 2 lbs.

Complaint:- (1) Progressive breathlessness on exertion for one week; (2) Breathlessness at night for one wk.

History:- Two years ago, the patient had high blood pressure, the exact figure was not known, for which he was admitted to another hospital, where sympathectomy was performed to lower the blood pressure. Prior

Prior to Sympathectomy, the patient had severe throbbing frontal headache on rising in the morning, lasting several hours - Duration one year, and intermittent in character. Headache was completely relieved after Sympathectomy.
Previous Illness :- Nil. Habit: Smoker of cigarettes.

Family History:- Mother died in 1917 of cerebral haemorrhage. Father died of Septicaemia. One brother and three sisters all alive and well.

On examination:- Robust, apparently healthy looking man of average intelligence. No prominent veins on the neck.
Cardiovascular System:- Pulse Rate: 84 per min., regular and vessel wall not palpable. B.P. 208/130 MM Hg. on admission. Heart:- Apex beat: 5th left intercostal space, one inch outside the Mid-clavicular Line. Auscultation:- 4th sound at the apex. Aortic 2nd sound accentuated.

Lungs:- Bilateral basal crepitations.

Investigations:- X-Ray of heart: Left ventricular enlargement and unfolding of aorta; E.C.G.: Left axis deviation. Sodium Amytal Test:- No change in Blood pressure. Blood Urea Nitrogen:- 18 mgm%.

Urine:- Total volume: 1660, Acid in reaction, sp.gr. 1020; albumin nil. Urea Range:- Concentrated specimen sp. gr. 1020; urea 3·5 gm%. Dilute specimen sp. gr. 1005; Urea 1·0 gm%. Fundal changes: Grade II.

Diagnosis:- Left Ventricular failure due to Essential Hypertension for which Sympathectomy was carried out two years ago.

Treatment:- (1) Bed rest for 4 weeks; (2) Aminophylline 0·24 Gm. I.V.I. at night; (3) Aminophylline 0·24 Gm. T.i.d. orally; and Phenobarbitone gr. ½ T.i.d. orally.

Result:- (1) Much improved; (2) Basal Crepitation disappeared; (3) The 4th heart sound disappeared. The Blood Pressure as the patient improved rose to 240/130 on discharge. Pulse Rate: 80 per min. and regular on discharge.
Remarks:- No headache complained of while the patient was in the Hospital. It was completely relieved after Sympathectomy.

Case 14:- Male, aged 40, Miner, Married, wt. 9 st. 5 lbs.
Complaint:- Bleeding from the nose 3 days.
History:- (1) Dyspnoea on exertion for 3 yrs; (2) Breathlessness at night for 3 weeks; (3) Headache for 6 mths. The patient was perfectly fit until 3 years ago, when he first complained of breathlessness on exertion, which progressively became worse. Occasional but severe throbbing frontal headache on rising in the morning, and

worse on straining at stool, lasting several hours. Duration 6 months. Had violent headache prior to Epistaxis, but since then the headache was completely relieved. No precordial pain.

Previous Illness: Nil.

Habit: Smoking. Family History:- Uneventful.

On Examination:- A healthy looking man, suffering from Epistaxis.

Cardiovascular System:- Pulse Rate: 100 per min, Irregular and thickened vessel wall. B.P. 220/110 on admission. Heart: Apex Beat:- 6th Intercostal space in Anterior Axillary Line. Auscultation: 4th heart sound at the apex. Aortic 1st and 2nd sounds accentuated. Lungs:- Bilateral crepitation.

Investigations:- (1) X-Ray: Left Ventricular enlargement with unfolding of the aortic arch; (2) E.C.G.: Left Axis deviation; (3) Blood Urea Nitrogen: 58 mgm%. (4) Urea Range:-

Conc. specimen sp.gr. 1018 - Urea 2 Gm.%.
Dilute specimen sp.gr. 1003 - Urea 1·0 Gm%.

(5) Urine: Total volume 2000 c.c., Reaction, acid. sp.gr. 1018. Trace of albumin and Hyaline casts present; (6) Hb. Estimation: 6% on admission; (7) Sodium Amytal Test: Not done; (8) Fundal Changes: Grade III.

Diagnosis: Essential Hypertension with left ventricular Failure.

Treatment:- Four days after his admission, the patient complained, in early morning at 6 a.m., of intense headache, followed by an attack of hypertensive encephalopathy; at 11 a.m. his blood pressure rose to 230/160 mm Hg. The attack was treated by 50 c.c. Sucrose (50%) intravaneously and the patient recovered.

Other Treatment:- (1) Rest in bed and "head-up" position when required; (2) Phenobarbitone gr. i T.i.d. for a week, then reduced to gr. ½ T.i.d. for a week; (3) Ferrous sulphate grs. iii T.i.d. to raise his haemoglobin level; (4) Aminophylline 0·2 Gm. T.i.d. for cardiac asthma.

Result:- (1) Blood pressure reduced to 200/110 on discharge; (2) Pulse Rate 74 per min. and Regular; (3) 4th heart sound still audible on discharge; (4) headache relieved.

Case 15:- Male, aged 47, gardner, married, wt. 10 st. 10 lbs.

Complaint:- (1) Breathlessness and precordial pain on exertion for 3 months; (2) Intermittent frontal throbbing headache for 3 months.

History:- Three months ago, the patient noticed increasingly shortness of breath on exertion, associated with sharp pain over the precordium. Throbbing frontal

headache on rising in the morning, lasting for 2-3 hours. Moderate in severity. Duration 3 months.

Previous Illness:- Mastoidectomy done while in Army.

Habit: Smoker and Drinker. Family History: Nil to note.

On examination:- A pleasant-looking man of average intelligence.
Cardiovascular System:- Pulse rate 80 per min., regular and vessel wall not palpable. B.P. 170/100 mm Hg. on Admission. Heart:- Apex beat: 5th Intercostal space, one inch outside Mid-clavicular Line. Auscultation: Accentuated 1st sound at the mitral area; 2nd aortic sound accentuated.

Lungs:- Bilateral basal crepitation.
Investigations:- (1) X-Ray of heart: Slight left Ventricular enlargement and slight unfolding of Aorta; (2) E.C.G.:- Left Axis deviation; (3) Urea Range: Con. specimen sp.gr. 1020 - Urea 2·6 Gm%.
Dilute specimen sp.gr. 1008 - Urea 0·6 Gm%.;
(4) Urine:- Total volume 1500 c.c., Acid reaction, sp.gr. 1020; albumin Nil; (5) Blood Urea Nitrogen:- 16 Mgm%; (6) Sodium Amytal Test: Blood Pressure fell from 170/100 to 140/95; (7) Fundal changes: Grade I.
Diagnosis:- Essential Hypertension.
Treatment:- (1) Rest in bed and "head-up" position when required; (2) Phenobarbitone gr. ½ T.i.d. for two weeks; (3) Pethidine 100 mgm S.c.i. relieved headache. Then (4) Pethidine 100 given orally.
Result:- (1) Symptoms relieved; (2) B.P. 150/100 on discharge; (3) Pulse Rate 80 per min. and Regular; (4) Headache relieved by Pethidine.

Chronic Glomerulo-Nephritis.

Case 16: Male, 45, Brick-layer, married, wt. 8 st. 8 lbs.
Complaint:-(1) Headache for one week; (2) Loss of speech for 4 days.
History:- Six years ago, the patient had an attack of Acute glomerulo-nephritis, and was informed after the attack was over, that there is still albumin in his urine. Nocturnal frequency of urination for the last 6 years. Began to suffer from frontal headache a week ago. It is throbbing and moderate in severity, occurring on and off during the day, lasting about ½ an hour. For the last 4 days, he has been unable to sleep due to headache, which also started on wakening in morning and felt sick. Recent complaint of defective vision.

Previous Illness:- Measles during childhood. Acute Nephritis 6 years ago. No history of Tonsilitis or "Sore Throats". Habit: Moderate drinker and smoker.

Family History:- Uneventful.
On Examination:- Very pale face with puffiness of eye-lids and face on admission.

Cardiovascular System:- Pulse Rate: 80 per minute, Regular and vessel wall not palpable. B.P. 160/100 on admission. Heart:- Apex beat: 5th intercostal space in Mid-clavicular line. Auscultation:- Heart sounds normal.

Renal System:- (1) No palpable tumour felt in both Renal region. Respiratory system: Nil to note.

Investigations:- (1) Urine:- Total volume 1500 c.c. on admission. Acid reaction. Sp. gr. 1016. Albumin +, R.B.C. +, W.B. C. + and Hyaline and granular casts present; (2) Blood Urea N_2:- On admission 82 Mgm%.
On discharge 54 Mgm%.
(3) Urea Range Test:-
Con. specimen: sp.gr. 1018 - Urea 3 Gm%.
Dil. specimen: sp.gr. 1002 - Urea 1 Gm%.;
(4) Co_2 combining power of Blood:- 54 vols.%.;
(5) Fundal examination revealed Grade III.

Diagnosis:- Acute exacerbation of chronic glomerulonephritis.
Treatment:- (1) Bed rest; (2) Diet; Initially 50 Gms of protein in the diet, which gradually increased to normal diet as the condition improved.

Result:- (1) Headache completely relieved;
(2) Urinary output increased, on Discharge,
Urine: sp.gr. 1012. Trace of albumin and one or two granular casts present; (3) B.P. 150/90 mm Hg.; (4) Pulse Rate: 80 per min and regular,

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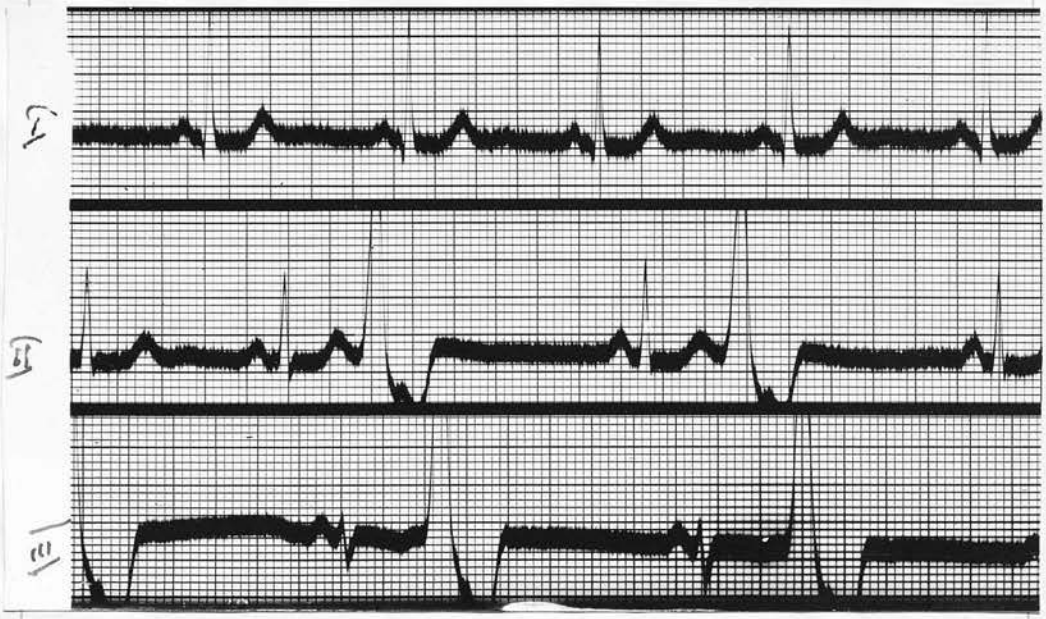
Case 17:- Female, 45, Housewife, Married, wt. 10 st.

Complaint:- (1) Dyspnoea on exertion for 3 days;
(2) Frontal and occipital headache for 3 weeks.

History:- The patient felt fit till 3 weeks ago, complained of moderate throbbing frontal and occipital headache on rising in the morning and on and off during the day, lasting several hours. Duration 3 weeks. During the attack, patient felt restless and slightly relieved by aspirin. The patient also noticed the swelling of her face and ankles in the morning during these three months, but did not complain of urinary disturbance. Four days before admission, the patient felt increasingly breathless on exertion.

Previous Illness: No history of scarlet fever; no history of Acute Tonsilitis or sore throat and no history of any kidney trouble. Menstrual history: Normal and no history of albuminuria of pregnancy.
Habit:- Nil. Family History:-Father died of Bronchitis; mother 82, died of "Stroke", children alive and well.

CASE N° 17



On Examination:- Very breathless and apprehensive lady.
Cardiovascular System:- Pulse rate 164 per minute, with multiple extra systoles, vessel wall not palpable. B.P. 180/100 MM Hg. on Admission.
Heart:- Apex beat: Not palpable. Auscultation:- Heart sounds not audible due to coarse crepitation on admission. After treatment for Pulmonary oedema - apex was localized in 6th intercostal space outside Mid-clavicular line, and heart sounds were closed.
Respiratory System:- Bilateral dulness over both bases with coarse crepitation.

Investigations:-(1) Urine: Total output 1500 - 1600 c.c. in 24 hrs. acidic, sp.gr. 1018, albumin ++, R.B.C. ++, Hyaline and granular casts +; (2) Urea Range:- Con.specimen - sp.gr. 1010 - Urea 1·5 Gm%.
Dil.specimen - sp.gr. 1008 - Urea 1·2 Gm%.;
(3) Blood Urea N_2 :- 63 mgm% on admission;
32 mgm% on discharge;
(4) E.C.G.: Left axis deviation with multiple ventricular extra-systoles; (5) Fundal Changes: Grade IV.; (6) Co_2 Combining power on admission 35 Vols.%
on Discharge ·50 vols.%.

Diagnosis:- Acute exacerbation of Chronic Glomerulo-Nephritis with left Ventricular failure and Pulmonary oedema.

Treatment:- (1) Bed rest; (2) Oxygen therapy; (3) Aminophylline 0·24 Gm. I.V.I., thereafter 0.24 T.i.d.; (4) Diet:- initially 50 Gms of Protein in the diet, which was gradually increased to normal as the condition improved.

Result:- (1) Pulmonary oedema cleared; (2) Headache completely relieved; (3) Urine:- sp.gr. 1011, trace of albumin and occasional granular casts; (4) B.P.: 150/90 mm Hg. on discharge. Pulse Rate:- 90 per min. and regular.

Case 18:- Male, aged 52, Weaver, Married.
Complaint:- (1) Diarrhoea, loss of appetite and vomiting for 2½ months; (2) Headache since a week.

History:- The patient was fit till 2½ months ago when he began to lose his appetite for all types of food and began to have attacks of hiccough after any food he ate. Felt sick, nauseating and vomited bile-stained gastric content, also had 2-3 watery motions a day. Since the last week, complained of severe throbbing frontal headache, which was constant, and worse in the morning, lasting several hours and unrelieved by medication.

Previous Illness:- Chicken-pox during childhood; malaria 1916; Dysentery 1917. No history of sore throats. Habit: Moderate smoker and drinker.

Family History:- Mother 60, died of cerebral Haemorrhage.

On examination:- pale, ill-looking man, breathing deeply with attacks of hiccough, dirty tongue with unpleasant odour to the breath.

Cardiovascular System:- Pulse Rate 90 per min, regular and vessel wall not palpable; B.P. 170/100 Mm Hg. on Admission.
Heart:- Apex beat:- 5th left interspace outside mid-clavicular line. Auscultation:- Heart sounds normal.

Lungs:- Bilateral dulness over both the bases of lungs and bilateral basal crepitation.

Investigations:- (1) Urine: Total volume in 24 hours: 1000 c.c. Acid Reaction, sp.gr. 1010, albumin, granular and Hyaline casts present. No R.B.C. and no W.B.C.; (2) Fundal Changes:- Grade IV. - Exudate and bilateral papilloedema; (3) Blood Urea N_2 : 116 Mgm%.; (4) Co_2 Combining power: 40 Vols.%.
Diagnosis:- Uraemia.
Treatment:- (1) Bed rest and Symptomatic; (2) 400 c.c. of 8% soda bicarb. drip intravaneously. Result: Died of Uraemia. Post Mortem Findings:- Bilateral contracted kidneys suggesting Chronic Glomerulo-Nephritis. Left Ventricular hypertrophy amd Pulmonary Oedema.

Case 19:- Female, 64, Housewife, Married, wt 14 st.
Complaint:- (1) Swelling of feet and face for 6 weeks; (2) Breathlessness on exertion for 5 years.

History:- Progressively increasing breathlessness on exertion for the last five years, also occasional frontal headache on rising in the morning, and aggravated by house-work. Moderate in severity, lasting about 2 hours, for which no medicine was taken.

Previous Illness:- Scarlet Fever at the age of 48. Pregnancies were normal. No history of albuminuria of pregnancy. Menopause at usual age. No history of sore throats. Family History:- Uneventful.
Habit: Nil. On Examination:- Obese lady with swollen face and legs.
Cardiovascular System:- Pulse Rate:- 100 per min. regular and vessel wall not palpable. B.P. 160/100 mm. Hg. on admission. Heart:- apex beat: Not palpable due to obesity. Heart sounds:- Faint, due to obesity. Lungs:- Normal.

Investigations:- (1) Urine: Total output 1200 c.c. in 24 hours on admission, Acid Reaction, sp.gr. 1016, albumin +, No R.B.C. amd no W.B.C. Hyaline and granular casts present; (2) Urea Range:-
Con. specimen - sp.gr. 1018 - Urea 2 Gm%.
Dil. specimen - sp.gr. 1012 - Urea 1·2 Gm%.;
(3) Blood Urea Nitrogen:- 118 mgm% on admission.
21 mgm% on discharge;
(4) Co_2 Combining power:- 44 Vols.%.

(5) Fundal Changes: Grade I.

Diagnosis:- Chronic Glomerulo-Nephritis.

Treatment:- (1) Bed Rest; (2) Diet:- Initially 50 gms. of Protein in the diet with low-salt intake. Protein gradually increased, but still kept on low salt diet with 1000 calories due to her obesity.

Result:-(1) The patient improved; (2) Blood Urea reduced to 21 mgm% on discharge; (3) Urine:- sp. gr. 1015, trace of albumin. B/P. 150/90 mm Hg. and Pulse Rate 80 per min. and regular. Headache relieved, oedema of face and feet cleared off.

Case 20:- Male, aged 68, retired, fine man. Married wt. 8 st. 6 lbs.
Complaint:- (1) Increasing breathlessness on exertion for 2-3 weeks; (2) Frontal headache since 6 months.

History:- Swelling of face in morning for 6 mths. Throbbing frontal headache on rising in the morning, lasting for about an hour, moderate in severity and felt sick during the attack, for which no medicine was taken.

Previous Illness:- Peptic Ulcer in 1940; No history of sore throats or Tonsilitis, and no history of kidney trouble. Habit:- Occasional smoker.
Family History:- Uneventful.

On Examination:- A pale man with swollen face.

Cardiovascular System:- Pulse Rate:- 90 per min, regular and vessel wall not palpable. B.P. 180/100 Mm Hg. on admission. Apex beat: 5th Interspace just outside mid-clavicular line. Heart sounds normal.
Lungs:- Few bilateral basal crepitation.

Investigations:- (1) Urine:- Initial output 800 c.c.; sp.gr. 1020, acidic, thereafter the urine output increased to 2500 c.c. sp. gr. 1016, albumin +, R.B.C.+, and granular casts +; (2) Blood Urea N_2
50 Mgm% on admission;
30 Mgm% on discharge;
(3) Co_2 combining power: 52 vols.%.; (4) E.C.G.:- Left axis deviation; (5) Fundal changes: Grade III.
(6) Urea-Range: con.specimen sp.gr. 1016 - 2.4 Gm%.;
dil.specimen sp.gr. 1010 - 1·2 Gm%.

Diagnosis:- Acute exacerbation of Chronic Glomerulo-Nephritis.
Treatment:- (1) Bed rest; (2) Low Protein diet of 50 gms., gradually increased to 60 gms., and then 75 gms. as the condition improved.
Result:- (1) Headache relieved; (2) B.P. 160/110 on Discharge; (3) Urea Nitrogen reduced to 30 mgm%. Oedema cleared off; (4) Urine: sp.gr. 1015, trace of albumin. No R.B.C. and occasional granular casts.

Case 21:- Male, aged 16, school-boy; wt. 9 st. 12 lbs.

Complaint:- Nil.
History:- On routine school-medical-examination found to have albuminuria and admitted to the ward for investigation. No Headache.
Previous Illness:- Scarlet Fever in 1943. No sore throat and no kidney trouble. Habit:- Nil.
Family History:- Uneventful.

On Examination:- Apparently healthy, well-built boy with no obvious disease. No oedema.

Cardiovascular System:- Pulse Rate:- 70 per min, regular and vessel wall not palpable. B.P. 150/90 mm. Hg. on admission. Apex Beat: in normal position. Heart sound:- closed. Lungs:- Nil to note.
No palpable tumour in the renal regions.

Investigations:- (1) Urine:- Total output 1500 c.c. in 24 hours, acidic, sp.gr. 1012, trace of albumin and occasional granular casts. No R.B.C.; (2) Blood Urea N_2:- 23 mgm% on admission; (3) Van Slyke-Urea-clearance Test:- 66% of the normal; (4) Fundal examination: Normal. No septic foci detected.
Diagnosis:- Chronic Glomerulo-Nephritis with no symptoms. Treat: Nil.

On Discharge:- Blood Urea Nitrogen and B.P. remains the same. Remark :- Nephritis resulting presumably from scarlet fever. No evidence of orthostatic albuminuria.

Case 22:- Male, 38, fitter, single, wt. 9 st. 7 lbs.

Complaint:-(1) Feeling of lethargy and tiredness for 1 month; (2) Constant pain behind the eyes since 4 days; (3) Severe occipital headache since a month.
History:- The patient was feeling well till a month ago; complained of lassitude and tiredness. Shortly after that he experienced severe throbbing occipital headache on wakening in the morning, lasting 1-2 hours, unrelieved by treatment - duration one month, and felt sick during the attack. Since last week noticed the swelling of the face in morning, and blurring of vision since 4 days. Had nocturnal frequency for the last 4 months.
Previous Illness:- Acute Nephritis 1933. No history of sore throats. Habit:- Smoker and drinker moderately.
Family History:- Nil to note.

On Examination:- Stout, well-built individual of an average intelligence; tendency to hurried breathing.

Cardiovascular System:- Pulse Rate: 80 per min. and vessel wall not palpable. B.P. 200/140 MM Hg. on admission. Apex-beat:- 5th interspace, within mid-clavicular line.

Auscultation:- Soft systolic murmur at the mitral area propagated into the left atria. Aortic 2nd sound accentuated.

Respiratory System:- Respiration: 30 per minute. No kidney tumour palpable in renal angle.

Investigations:- Urine:- Total output 3000 c.c. in 24 hours, acidic, sp.gr. 1010, albumin ++, R.B.C. ++, Blood Casts + and granular casts present. Blood Urea Nitrogen:- 155 mgm%. Co_2 combining power: 24 vols%. Fundal Changes:- Grade IV. - Disc blurred, silver-wiring of the arteries, small flame-shaped haemorrhage and very many exudate. E.C.G.: Left axis deviation. Blood Creatinine:- 7.5 mgm%.

Diagnosis:- Chronic Glomerulo-Nephritis and Uraemia.

Treatment:- (1) Bed rest; (2) Only Symptomatic. To relieve headache, Sodium Amytal grs. iii. once and aspirin grs. x. when required.
Result:- Died of Uraemia. No Post-mortem done.

Case 23:- Male, 39, Foreman, Married, wt. 11 st. 5 lbs.

Complaint:- (1) Tiredness for 3 months; (2) Swelling of the face in morning for a week; (3) Nocturnal frequency of urination since 6 months; (4) Severe throbbing frontal headache on wakening in morning, lasting several hours, unrelieved by treatment, and feeling sick during the attack - duration 3 months.

Previous Illness :- Nil to note. Habit:- None.
Family History:- Uneventful.

On examination:- Well built intelligent person.

Cardiovascular System:- Pulse Rate:- 90 per min., regular and vessel wall not palpable. B.P. 240/140 mm. Hg. on admission. Apex beat:- 5th left inter-space ½ inch outside the mid-clavicular line. Second aortic sound accentuated. Respiratory Rate 25 per minute, and few bilateral basal crepitation. Both kidneys not palpable.

Investigations:- Urine: Total output 2000 c.c. in 24 hours, acidic, sp.gr. 1010, albumin +++, R.B.C. Nil. Granular and Hyaline casts present. Urea Range:-
Con. specimen sp. gr. 1010 - Urea 1 gm%.
Dil. specimen sp. gr. 1010 - Urea 0·5 Gm%.
Blood Urea Nitrogen:- 121 mgm%. Blood Creatinine: 6 mgm%. Co_2 Combining power : 48 vols%.
Fundal Changes:- Blurring of discs, with woolly exudate, few flame-shaped haemorrhages, and narrowing of vessels (Grade IV.)

Diagnosis:- Chronic Glomerulo-Nephritis with Uraemia and terminal broncho-pneumonia.

Treatment:- (1) Bed rest; (2) Symptomatic. To relieve headache Sod. Amytal grs. iii. and Aspirin gr. x. given without any effect. Result:- The patient died.
Post Mortem Findings:- Chronic Glomerulo-Nephritis Left Ventricular Hypertrophy and terminal Broncho-Pneumonia.

Case 24:- Female, 53, Housewife.
Complaint:- (1) Drowsiness and unwell for 6 weeks; (2) Constant, severe throbbing frontal headache worse in the morning, lasting several hours, and unrelieved by any sedation; duration 6 weeks. No other history obtainable from the patient.
On Examination:- Well-built, well-nourished middle-aged lady.
Cardiovascular System:- Pulse rate: 90 per min., regular and vessel wall palpable. B.P. 200/150 Mm. Hg. on Admission. Apex Beat:- 5th left interspace, just outside mid-clavicular line. Pericordial friction rub heard over the sternum. Respiratory rate 30 per minute, and few bilateral basal crepitations.
Investigations:- (1) Urine: Total output 1500 c.c. on admission in 24 hours, acidic, sp.gr. 1013, albumin +, R.B.C. few, granular casts ++. Fundal Changes:- Bilateral papilloedema, exudate and occasional haemorrhages, vessel wall narrowed (Grade IV.); (2) Co_2 Combining Power:- Not done. (3) Blood Urea Nitrogen: 200 mgm%; (4) Urea Range:- Not done.

Diagnosis:- Chronic Glomerulo-Nephritis, terminal Uraemia and Pericarditis. Treatment: Symptomatic.

Result:- The patient died 2 days after admission.

Post-Mortem Findings:- Left Ventricular Hypertrophy chronically contracted kidneys and terminal pericarditis.

Case 25:- Male, 46, miner, married, wt. 9 st. 3 lbs.
Complaint:- (1) Headache and dizziness for 6 weeks; (2) Nausea and Vomiting for 1 week; (3) Nocturnal frequency of Micturation for the last year.

History:- The patient was well till six weeks ago, when on wakening in the morning felt throbbing frontal headache, associated with feeling of nausea and dizziness. The attack lasted about an hour and moderate in severity. The headache relieved after getting up without medication. For the last week, the patient felt nauseated and vomited after food.

Previous Illness:- Scarlet Fever at the age of 6. No history of sore throats. Habit:- Moderate smoker and drinker. Family History:- Uneventful.

On Examination:- Thin-built man of intelligence.

Cardiovascular System:- Pulse rate: 74 per minute, regular and vessel wall not palpable. B.P. 160/100 mm. Hg. on admission. Apex-Beat: 5th left intercostal space within Mid-clavicular line.
Auscultation:- 2nd aortic sound accentuated.

Investigations:- Urine:- Urinary output 2500 c.c. in 24 hours, acidic sp. gr. 1010, albumin +, R.B.C. Nil, few granular casts. (2) Urea Range:-
Con.specimen sp.g.r 1016 - Urea 2·5 Gm%.
Dil.specimen sp.gr. 1010 - Urea 1·2 Gm%.;
(3) Blood Urea Nitrogen:- 50 mgm. on admission;
30 mgm. on discharge;
(4) Fundal Changes:- Grade III; (5) E.C.G.:- Left Axis deviation.

Diagnosis:- Chronic Glomerulo-Nephritis.
Treatment:- (1) Bed rest; (2) Diet of 50 Gms. of Protein initially gradually increased as the patient improved.
Result:- Headache relieved and patient improved on discharge. Urine:- sp. gr. 1015, trace of albumin. B.P. 140/90 MM. Hg. and Pulse Rate 64 per min. and regular. Blood Urea Nitrogen 30 Mgm%. on discharge.

Case of Chronic Pyelo-Nephritis.

Case 26:- Female, 44, Housewofe, married.
Complaint:- (1) Passing of blood in urine and pain in right loin for seven months; (2) Severe frontal headache since the last 2 months; (3) Poor eyesight since 3 months.

History:- Ten years ago,the patient suffered from left renal colic and was found to have left-sided hydro nephrosis. Since then passing increasing amount of urine and for the last seven weeks, it is blood stained. Histerectomy for fibroid performed 12 years ago. Habit:- Nil. Family History:- Uneventful.
Physical Examination:- Pale, ill-looking thin woman. No oedema. Temperature 100°F.

Cardiovascular System:- Pulse Rate:- 82 per min., regular and vessel wall palpable. B.P. 230/150 MM. Hg. on admission. Apex Beat:- In 6th left Intercostal space outside mid-clavicular line.
Heart Sounds:- Normal. Lungs:-Congestion of both Lungs.
Investigations:-(1) Urine Examination:- Total output 2000 c.c. in 24 hours. Reaction acid, sp.gr. 1010, albumin +, Pus cells ++, B.Coli and R.B.C. present. Sugar nil; (2) Blood Urea Nitrogen: 28 mgm%. which raised to 86 mgm% just before death.

Cystoscopic Examination:- Cystitis, deformity of calicyces of both kidneys (indicative of old fibrosis).
Fundal Changes:- Grade III.
Diagnosis:- Chronic Pyelo-Nephritis.
Treatment:- (1) Bed rest; (2) Fluids; (3) "Sulpha Triad" for B.Coli. (Sulphathiazole, Sulphadiazine and Sulphamethazine); (4) Sedatives: Morphine gr. ¼ during uraemic stage; Result:- Died of Uraemia.

Post-Mortem findings:- (1) Congenital Hypoplastic left kidney with hydro-ureter and hydronephrosis; (2) Both chronic pyelonephritis. Arteriolosclerosis and Hypertensive hypertrophy of left ventricle.

Cases of Amyloidosis.

Case 27:- Female, 50, Housewife, Married, wt. 6 st. 8 lbs.
Complaint:- (1) Abdominal pain for 2 months;
(2) Vomiting of Blood for 5 hours.

History:- Three months ago, the patient began to have typical symptoms of duodenal ulcer, e.g. epigastric pain, relieved by taking food. Then the sudden massive haemetemesis lasting nearly five hours.

Previous Illness:- Rheumatoid Arthritis for the last 18 years. Menopause at usual age.

On Examination:- A very pale thin woman with large sucking pads on her cheeks and with very severe Rheumatoid Arthritis, affecting all the joints of the arms and legs.

Cardiovascular System:- Pulse Rate:- 100 per min., regular and vessel wall not palpable. B.P. 90/60 on admission after severe haemetemesis. Apex Beat:- 5th left interspace within mid-clavicular line. Heart-sound - closed. Lungs:- Bilateral basal crepitation. Liver:- enlarged - two fingers breadth below the right costal margin. Spleen:- Grossly enlarged, extending down to the umbilicus.

Investigations:- (1) X-Ray after Barium Meal:- Gross deformity of duodenal cap; (2) Stool:- Benzedine positive for first five days; (3) Hb. Estimation:- 30% on admission; (4) R.B.C. Count: 3½ million per c. mm. (5) Urine Examination:- Acidic, sp.gr. 1020, albumin +. No cast; (6) Fundal Changes: Normal. (7) Blood Sedimentation Rate:- 42 min. in one hour. (8) W.R. - Negative Congo-red Test - Positive. E.C.G.:- Left axis deviation.

Diagnosis:- Rheumatoid Arthritis, Amyloidosis and Duodenal Ulcer.

Treatment:- (1) Bed rest; (2) Blood Transfusion 2½ pints, and Blood Pressure rose to 170/90 and Hb. raised to 35%., thereafter rose steadily by Ferrous Sulphate; (3) Phenobarbitone gr. ½ T.i.d. for 2 weeks; (3) Mag. Trisilicate i. B.i.d.; (5) Ulcer diet. After one week: Ferrous Sulphate grs.iii. T.i.d.

The patient had frontal throbbing headache worse in the morning and on raising the head.

Result:- The Haemetemesis caseed and patient much improved and discharged after 2 months. B.P. rose to 150/100 on discharge and pulse rate 80 per min. and regular.

Case 28:- Female, 25, Bus-conductress; married; too ill to weigh.
Complaint:- (1) Diarrhoea and vomiting for 2 weeks; (2) Coughing since childhood; (3) Frontal headache for 2 weeks; (4) Nocturnal frequency of urination for 4 months.
History:- Since the age of 3, following an attack of pneumonia, the patient had persistent cough which brought up large quantity of foul-smelling sputum, especially in morning, and the patient became progressively breathless. Four months ago, following an attack of "Cold", she noticed the swelling of her face and both ankles in the morning. The patient experienced severe throbbing frontal headache, worse on rising in the morning and aggravated by coughing and vomiting, lasting about an hour - duration 2 weeks. Nocturnal frequency of micturation for 4 months. For the last fortnight, the patient had very severe watery diarrhoea associated with sickness and vomiting for 2 days.
Previous Illness:- Pneumonia at the age of 3; then frequent attacks of cold off and on. Menstruation: was normal till 3 months ago and stopped completely.
Family History:- Uneventful.
On Examination:- Pale, ill-looking woman, dyspnoic at rest, temperature 99°F on admission.
Respiratory System:- Respiration rate: 30 per minute, Bilateral finger-clubbing present. Lungs:- Bilateral dulness over the bases. Bronchial breathing over the left base, and coarse crepitation over both bases.

Cardiovascular System:- Pulse Rate:- 130 per minute, regular and vessel wall not palpable. B.P. 150/100 Mm. Hg. on admission. Apex-beat:- 5th left interspace within mid-clavicular line. Heart sounds: Normal. Liver:- enlarged, 2 fingers breadth below the right costal margin; spleen: Just palpable.

Investigations:- X-Ray of chest :- Opacity of left costo-phrenic angle suggesting effusion. Prominent Broncho-vesicular markings in both lower lobes associated with degree of lower lobe collapse on both sides.

Heart shadow within normal limit; (2) E.C.G.:- Normal. (3) Wassermann Reaction:- Negative; (4) Sputum:- Negative for Tuberclebacilli; (5) Blood Urea Nitrogen: 50 Mgm%.; (6) Fundal Changes:- Grade III. (Many soft exudate with narrowing of the artery); (7) Blood Sedimentation Rate:- 45 min. in one hour; (8) Urine:- Acid Reaction, sp.gr. 1015, albumin + with granular casts.
Diagnosis:- Bilateral Bronchiectasis with Amyloidosis.

Treatment:- (1) Rest in bed; (2) Sod. Amytal grs. iii to secure sleep; (3) Kaolin grs. xxx T.i.d. to check diarrhoea. During the patient's stay in Hospital, her condition deteriorated; she became Uraemic and complained of constant dull occipital headache, associated with vomiting and the patient died.
Post Mortem Findings:- (1) Diffuse bilateral bronchiectasis; (2) Amyloid disease of Kidneys, Liver, Intestine, Pancreas

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Case 29:- Male, 28, motor-driver, married, wt. 8 st.3 lbs.
Complaint:- (1) Dyspnoea on exertion and constant cough for the last 8 months.
History:- Pneumonia 12 years ago, since then, attacks of cough every winter. Recently, the cough has been constant and productive with large quantity of muco-purulent foul-smelling sputum. Loss of weight during the last 3 months. No complaint of headache. Previous Illness:- Pneumonia at the age of 9 and 12. Habit:- Smoker.
Family History:- Wife aged 26, alive and well. Parents alive and well.
On examination:- Pale, somewhat dyspnoic and spasm of coughing during physical examination. Temperature 99·5°F on admission.

Respiratory System:- Respiration rate 30 per min. marked finger-clubbing. Dulness on both bases, Broncho-vesicular breath sounds with coarse crepitations over both bases.
Cardiovascular System:- Pulse rate 100 per min, regular and vessel wall not palpable; B.P. 160/100 mm. Hg. on admission. Heart sound - Normal. Spleen and Liver just palpable.

Investigations:- (1) X-Ray of the chest:- Bilateral Basal Bronchiectasis; and heart shadow within normal limit; (2) E.C.G.: Normal; (3) Sputum for tubercle bacilli, negative; (40 Blood examination: R.B.C. count 4 million per c. mm. Total W.B.C. 11,000 per cmm., 70% Polymorphs; (5) Hb. Estimation:- 60% on admission; (6) Urine Examination: Acidic, sp.gr. 1014, albumin +, and granular casts present; (7) Blood urea Nitrogen: 60 mgm%.; (8) Fundal changes: Normal; (9) Congo-red Test - Positive for Amyloidosis.

(10) Water-dilution test:- Took over 4 hours to excrete 2 pints of fluid and sp.gr. fixed at 1914.
Diagnosis:- Bilateral Bronchiectasis, Amyloidosis and Anaemia.
Treatment:- (1) Bed rest; (2) Nourishing diet; (3) Postural drainage; (4) Penicillin Inhalation; (5) Liver injection in order to help, but failed to improve the blood picture (4 c.c. Anahaemin 1·M·1. twice a week - total 8 injections; (6) Ferrous Sulphate grs. iii. T.i.d.

Result:- Discharged with much improvement. Hb.:- raised to 85%; B.P. 160/100; Pulse 90 per min. and regular. Temperature normal on discharge.

Cases of Subarachnoid Haemorrhage.

Case 30:- 25, slater, unmarried, wt. 10 st. 7 lbs.
Complaint:- Sudden throbbing frontal headache, bursting, intense and progressive was associated with vomiting, which did not relieve the headache. Duration - 12 hours.
Previous illness:- Nil. Habit:- Smokes cigarettes.
Family History:- Uneventful.

On Examination:- A well-built man lying curled up in bed in a semi-conscious state. Temperature 99·4. Respiration 24 per minute.
C.N. System:- Rigidity of the neck; both pupils dilated, but reacted to light and accommodation. Positive Kernig's, and absence of sensory motor disturbance. Reflexes are present.

Cardiovascular System:- Pulse rate 72 per minute, regular and vessel wall not palpable. B.P. 140/100 Hg. on admission. Apex beat:- left 5th space within Mid-clavicular line. Heart sound, normal.

Investigations:- Diagnostic L.P. removed 5 c.c. of C.S.F.; Blood-stained C.S.F., Pressure 200 mm. H_2O; Protein 70 mgm%; R.B.C. Loaded; sugar 85 mgm%.; Chloride 680 mgm%., W.R. Negative.
Fundal Examination:- No Papilloedema. Urine, Normal.
Diagnosis:- Spontaneous Subarachnoid Haemorrhage.
Treatment:- (1) Bed rest; (2) Pethidine 100 mgm. 1·M·1. (3) Lumbar puncture on two occasions and removed 5 c.c. of C.S.F. on each occasion. The Lumbar puncture showed the pressure coming down and C.S.Fluid becoming clear.
Result:-The patient discharged as symptom free; Frontal headache completely relieved; Pulse Rate 70 per minute and regular. B.P. 120/80 mm. Hg. on discharge. No sign of any paralysis.

Case 31:- Female, 58, Housewife, married, wt. 9 st.12 lbs.

Complaint:- Bursting Headache of sudden onset, distributed all over the head associated with vomiting.
Duration of several hours.

History:- The patient experienced suddenly, a severe bursting headache all over her head - both front and back - fell to the floor, but didn't loose consciousbess. She lay on the floor for a minute or two, then picked herself up and went to bed.
Headache was terrible, persisting all the time. Her head felt dizzy. The headache persisted all over the head, but seems to have been caused by shutting her eyes. The light does not cause her pain. Moving her head aggravated the headache, especially on bending her head forward.

Previous Illness:- (1) Only slight tingling headache over the crown and the brows; (2) For the last nine years has had a tendency to frontal headache and fatigue. Menopause at usual age.

Family History:- Husband aged 60, alive and well. Daughter aged 20, alive and well. Father died at 71, of cerebral haemorrhage; mother died at 79 of cerebral haemorrhage. Sister died suddenly at 58, due to Heart Attack ?

General Examination:- A stout, elderly lady with good colour; fully conscious. Temperature 100°F on admission; Respiration 25 per minute.

C.N. System:- Slight rigidity of neck muscles; Pupils equal and reacted to light and accommodation. No loss of motor power of limbs; speech unaffected. No loss of sensation in the face; no facial weakness. All reflexes are present and equal; both planter-flexor present.

Cardiovascular System:- Pulse Rate 52 per minute, regular and vessel wall not palpable. B.P. 180/90 mm. Hg. on admission. Apex Beat:- 5th left intercostal space just outside mid-clavicular line. Heart sound:- closed.

Investigations:- Diagnostic Lumbar puncture and removed 5 c.c. of c.s.fluid. C.S.F. blood-stained; Pressure 200 mm. H_2O; Protein 80 mgm%.; R.B.C.: Loaded; Caloride 720 mgm%. Glucose 80 mgm%. W.R. Negative; (2) Fundal Exm.:- Normal; (3) Urine exm.:- Nothing to note.

Diagnosis:- Spontaneous Subarachnoid Haemorrhage.

Treatment:- (1) Bed rest; (2) Luminal gr. i. B.i.d. for 3 weeks; Aspirin grs. x R.i.d.; (3) Therapeutic lumbar puncture on two occasions removing 5 c.c. c.s.f. on each occasion.

Result:- Discharged symptom-free. Headache relieved. B.P. 160/90 mm. Hg. Pulse rate 80 per min. and regular.

Case 32:- Female, 75, Housewife, married, wt. 10 st. 5 lbs.
Complaint:- Difficulty in swallowing food and liquid for 3 days. Sudden bursting frontal headache of 3 days duration.
Previous Illness:- None. Menopause at usual age.
Family History:- Mother died at 60 of Hypertension. Father died at 65 of cancer of stomach. Husband, aged 70, died of Pneumonia. Children three, all alive and well.
On examination:- An elderly lady, looks well, and not unconscious. Temp. 97°F. Respiration 22 per minute.
C.N. System:- No sign of rigidity of the neck; Pupils equal and reacted to light and accommodation. Reflexes in both limbs present and equal. Bilateral Planter flexor response present.
Cardiovascular System:- No other sensory or motor disturbance. Pulse Rate:- 72 per minute. Regular and vessel wall palpable. B.P. 190/100 mm. Hg. on admission. Apex Beat:- 5th left intercostal space just outside mid-clavicular line.
Heart Sounds:- 2nd Aortic accentuated.

Investigations:- Diagnostic Lumbar Puncture and removed 10 c.c. C.S.F. Blood-stained. C.S.F. Exm.:- Pressure:- 300 mm. H_2O; Protein 80 mgm%.; R.B.C. Plenty ; Chloride - 700 mgm.%. glucose 80 mgm%. W.R. Negative. Urine:- Normal.
Fundal Exm. :- Grade I.
Diagnosis:- Spontaneous Subarachnoid Haemorrhage.

Treatment:- (1) Bed rest; (2) Aspirin grs. x. T.i.d. for 4 days; (3) Diagnostic L.P. once.
Result:- Discharged symptom-free. Headache relieved by diagnostic lumbar puncture once, and aspirin eased headache a little. Difficulty in swallowing relieved. B.P. 180/100 MM. Hg. Pulse Rate 72 per min., and regular. Temp. 97°F.

Case 33:- Female, aged 56, Housewife, married, wt. 11 st. 2.
Complaint:- Sudden unconsciousness and on recovery severe bursting frontal headache with vomiting.
Previous Illness:- Operated for uterine prolapse 30 years ago. Menopause at usual age.
Family History:- Husband 56, suffering from Pernicious Anaemia.
On examination:- Unconscious on admission, but responded to painful stimuli. Temp. 99°F. Respiration, 22 per min.
Central Nervous system:- Slight rigidity of the neck muscles; pupils small and contracted, but reacted sluggishly to light. Deep tendon-reflexes are absent in all limbs. Both extensor response present. Sensory changes could not be tested, all limbs are flaccid.
Cardiovascular System:- Pulse rate 40 per min, regular and vessel wall not palpable. B.P. 160/100 mm. Hg. on admission. Apex beat:- In 5th left Intercostal space outside mid-clavicular line.
Heart sounds: Normal.

Investigations:- Diagnostic L.P. removed C.S.F. Blood stained, 5 c.c. C.S.F:- Blood stained; Pressure 320 mm H_2O. Protein 80 mgm%. R.B.C. Plenty. Chlorides 710 mgm%. Glucose 78 mgm%. W.R. Negative.
Fundal Exam.:- Normal.
Diagnosis:- Spontaneous Subarachnoid Haemorrhage.
Treatment:- (1) Bed rest; (2) Therapeutic L.P. performed once removing sufficient C.S.F. to bring the pressure down, which relieved headache. Aspirin grs. x. T.D.S.
Result:- Discharged as symptom-free. Headache relieved by Lumbar Puncture. B.P. 160/90 mm. Hg. Aspirin did not relieve the headache. Pulse Rate 80 per min. and regular. Both planter flexor and no residual paralysis. All reflexes returned to normal.

Case 34:- Female, aged 48, House-wife, married.
Complaint:- Sudden severe, throbbing and bursting right temporal headache with vomiting for 2 days.
History:- None. Menstruation stopped 2 years ago.
Family History:- Uneventful.
On examination:- The patient was fully conscious on admission. Temperature 100°F. Respiration 20 per min.
Central Nervous System:- Rigidty of the neck muscle; Positive Kernig's; pupils equal and reacted to light and accommodation. All reflexes are exaggerated in limbs. Plantar response both extensor.
Cardiovascular System:- Pulse rate 75 per min. Regular and in time and force. Wall not palpable. B.P. 150/100 mm. Hg. on admission. Apex Beat:- In 5th left Intercostal space within mid-clavicular line. Heart sound closed.
Investigations:- Diagnostic Lumbar puncture and removed C.S.F. to relieve pressure. C.S.F. Blood-stained. Pressure 250 mm. H_2O; Protein 150 mgm%. R.B.C. Plenty; Chlorides 700 mgm%. Glucose 76 mgm%. W.R. Negative.
Urine Exm.:- Normal. Fundal Findings:- Normal.
Diagnosis:- Spontaneous Subarachnoid Haemorrhage.
Treatment:- (1) Bed rest; (2) Diagnostic Lumbar Puncture;on one occasion. Six days after admission, the patient suddenly collapsed and died.
Post Mortem findings:- Subarachnoid Aneurysm and Circle of Willis.

Cases of Cerebral Tumours.

Case 35:- Male, 49, Butcher, Married, wt. 7 st. 12½ lbs.

Complaint:- (1) Generalised convulsive fits preceded by headache over the left eye; (2) Headache over the right eye, sharp and constant in character, relieved after the attack of fits. No history of vomiting. Duration - 4 weeks.
Previous Illness:- Acute Glomerulo-Nephritis at the age of 15. Family History:- Nil to note.

General Exm.:-Intelligent and co-operative despite dysarthria.
Central Nervous System:- Pupils small and equal, reacted to light and accommodation. Sense of smell present. Absence of occular palsy; No precipitate visual defect. All reflexes are exaggerated on the right side.
Cardiovascular System:- Pulse rate 50 per minute;wall not palpable. B.P. 170/70 mm Hg. on admission; Apex Beat:- 5th left interspace within mid-clavicular line. Heart Sounds:- Normal.

Investigations:- 1. C.S.Fluid: Pressure 100 mm H_2O. Protein 100 mgm potent; R.B.C. Nil; W.B.C. 3 per cmm., Chloride 718 mgm per cent; glucose 72 mgm per cent.; (2) Fundal examination: Blurring of vision at the nasal margin and bilateral papilloedema; (3) Urine exm.:- Nil to note; (4) Ventriculography:- filling defect in left lateral ventricle; (5) Electro-encephalography: Moderate dysrythmia in left mid-parietal region.
Diagnosis:- Left-sided cerebral tumour.
Treatment:- Operated and found glioma involving left frontal area.
Result:- Died 3 days after operation.

Case 36:- Male aged 32, Mechanical Engineer, married, wt. 9 st. 13 lbs.
Complaint:- (1) Jacksonian epileptic fits starting in the left arm and becoming generalised since 8 months; (2) Headache coming on before the fits, site of headache, left temporal side, constant and dull in character. No associated symptoms and no history of aura.
Previous Illness:- No history of trauma and no history of Epilepsy in the family. Habit:- Nil.
Family History:- Not relevant.

On examination:- A healthy looking man with no obvious disease.
Cardiovascular System:- Pulse rate 80 per min. regular, vessel wall not palpable. B.P. 170/90 mm. Hg. on admission. Apex beat:- 5th left space, within mid-clavicular line. Heart sounds: Closed.

Central Nervous System:- Pupils equal in size and shape; reacted to light and accommodation. No sign of ocular paralysis. Sense of smell present; no disturbance of vision; all reflexes are present and exaggerated with no localizing sign.

Investigations:- (1) C.S.Fluid. Pressure 120 mm H_2O. Protein 120 mgm%; W.B.C. 4 per cmm. R.B.C. None; chloride 716 mgm%. glucose 69 mgm%. W.R. Negative; (2) X-Ray of skull:- Calcification and increased vascular markings in right temporal zone of skull; (3) Electro-encephalogram:- Localised dysrythmia over the right fronto-temporal region. Fundal Exm.:- Normal. Urine exm.:-Nil to note.

Diagnosis:- Meningioma.
Treatment:- Surgically removed.
Result:- The patient discharged as cured. Headache - disappeared. B.P. 140/80 mm. Hg. Pulse rate 80 per minute and regular.

Case 37:- Male, 58, Iron-worker, married, wt.13 st.4 lbs.
Complaint:- (1) Change in personality for the last 6 months. No history of headache.
Previous Illness:- Nil. Habit:- Nil.
Father History:- Father died at 72 of cerebral haemorrhage. Mother died at 84 yrs. of heart-failure; Children aged 22, 15 and 12 alive and well.
On Examination:- An un-coperative man with defective memory, with Jargon Aphasia.

Central Nervous system:- Pupils equal in size and shape and reacted to light and accommodation; (2) Blurring of nasal margin of the Disc. No sign of ocular paralysis. Sense of smell present; (3) Light Homonymous Hemianopsia. Cortical sensory loss of right side of the body. Paralysis of right arm and right leg of upper motor neurone type. Right knee jerk and right ankle jerk exaggerated. Left arm and left leg; normal reflexes and left plantar flexor response present.

Cardiovascular system:- Pulse rate: 90 per min., regular and vessel wall not palpable. B.P. 170/100 mm Hg. on admission. Apex Beat:- Within normal limits. Heart sound normal.
Investigations:- (1) C.S.Fluid: Pressure 200 mm. H_2O. Protein 80 mgm%.; W.B.C. None; R.B.C. None; Chloride 710 mgm%.; glucose 85 mgm%.; W.R. negative. (2) Fundal Exm.:- Bilateral papilloedema; (3) X-Ray pf Skull:- Nil to note; (4) Urine exm.:- Nil to note. (5) Ventriculography: Both lateral ventricles grossly dilated. A large mass causing upward displacement of Posterior half of the floor of the left lateral ventricle. This compressed the proximal part of temporal horn and displaced it laterally.

Displacement of 3rd ventricle to the right.
Diagnosis:- Cerebral Tumour.
Treatment:- Surgically attempted.
Result:- Died.
Histologically:- A large left-sided malignant Astrocytoma was found.

Case 38:- Male, 48, Boiler-maker, wt. 7 st. 7 lbs.
Complaint:- (1) Paralysis of left arm and hand since 4 weeks; (2) weakness of left leg since 2 weeks ; (3) Occipital headache for the last 3 weeks. Headache worse in the morning and at night. Constant in character. No associated symptoms and unrelieved by treatment. Previous History:- Nil.
Habit:- Smokes cigarettes.
Family History:- Nil to note.
General exm.:- Drowsy and un-coperative, disorientated in time and space.
C.N. System:- Pupils:- reacted to light and accommodation. Peripheral Vision examination was not carried out as patient could not co-operate.
Tests for Sensory distribution not obtained due to non co-operativeness of the patient.
Reflexes:- Left extensor response present. All reflexes are exaggerated on left side of the left leg.

C.V. System:- Pulse rate:- 70 per min., regular and vessel wall not palpable. B.P. 150/110 mm Hg on admission. Apex beat:- 5th left interspace within mid-clavicular line. Heart sounds closed,

Investigations:- C.S.Fluid:- Pressure 150 mm H_2O. Protein 65 mgm%.; W.B.C. 1 per cmm. R.B.C. None. Chloride 660; glucose 69; W.R. Negative.
Fundal Exm.:- Bilateral papilloedemia.
Urine Exm.:- Nil to note.
Diagnosis:- Cerebral Tumour.
Treatment:- Symptomatic.
Result:- Died.
Post-Mortem findings: Right-sided glioblastoma.

Cause of Cushing's Syndrome.

Case 39:- Female, 31, Housewife, married, wt. 17 bstone.
Complaint:- (1) Admitted for intermittent Biliary colic since one year; (2) Getting very fat and also beard for the last 8 years; (3) Since the last 8 years, occasional throbbing temporal and frontal headache, worse in the morning, aggravated by vomiting, and relieved by rest; (4) Amenorrhoea for the last 3 years. Previous Illness: Nil to note.
Family History:- Father died of accident at the age of 40; Mother aged 60 alive and well; no children.

On examination:- Very obese; hirsutes with moustache, and purple striae over the lower abdomen.
Cardiovascular System:- Pulse rate: 72 per min.; regular and vessel wall not palpable; B.P. 145/100 mm. Hg. on admission. Heart sound:- faint,but closed.

Investigations:- (1) Fundal examination: Nil to note. (2) Field of vision;- normal; (3) Urine: Normal; (4) X-Ray of gall bladder: Non-functioning gall bladder with gall stones; (5) Blood examination:- R.B.C. count: 5·2 millions per c. mm.; (6) Hb. estimation: 110%; (7) Ketosterosis: Not estimated; (8)
Diagnosis:- Cushing's Syndrome.

Treatment:- Treated for Chronic Cholecystitis, (1) 1000 calories diet; (2) Thyroid gr. i. B.i.D. On dietetic treatment, the weight reduced to 15 stones and 6 lbs. Then she was operated upon and removed chronically infected gall bladder - containing two gall-stones.
Result:- Headache relieved. B.P. 150/100 mm. on discharge.

Case 40:- Female, aged 53, Secretary, Unmarried, wt. 16 st. 10 lbs.
Complaint:- (1) Polyuria and Pruritis Vulvae for the last 3-4 months; (2) Throbbing frontal headache worse in the morning and aggravated by straining at stool, and relieved by rest and by avoiding constipation. Duration - 6 months.
History:- Tended to be fat from the age of 12. Since the age of 16, hair appeared on her chin, and striae on her abdomen. First menstruation at the age of 13, but her menstruation was most irregular ever since.
Previous Illness:- Thread-worms during childhood.
Family History:- Father aged 68; mother aged 63, both alive and well.
On examination:- Very obese, hirsutes and purple striae over the abdomen.
Cardiovascular System:- Pulse rate 82 per min., regular and arterial wall not palpable. Apex Beat:- Not palpable. Heart sounds:- Normal.

Investigations:- (1) Fundal exam:- Slight "silver-wiring of the retinae artery; (2) Field of vision: Normal; (3) Urine:- Sugar +; (4) Fasting blood sugar 194 mgm%.; (5) Blood Exm.:- R.B.C. count: 5·5 million per c.mm. Hb. estimation: 109%.; (6) Ketosteroids: Not estimated.
Diagnosis:- Cushing's Syndrome.
Treatment:- (1) Dietetic 1600 calories with 130 gms. of Carbohydrate. This reduced to 1000 calories with 100 grams of carbohydrate.
Result:- The patient became sugar free on diet only, and discharged symptom-free. The pruritis vulvae relieved by controlling glucosuria.

Cases of Menopausal Disturbance.

Case 41:- Female, 48, housewife, married, wt. 9 st.4 lbs.
Complaint:- Throbbing frontal headache, worse on rising in the morning, lasting about half an hour, associated with flushing and sweating. Moderate in severity. Duration - 3 months.
Previous Illness:- Gastric ulcer 12 years ago. Menstruation stopped six months ago. Before that, it was quite regular. No history of dymenorrhoea. All pregnancies were normal. Family History:- Not relevant.
On examination:- A thin and rather depressed looking woman. Cardiovascular System:- Pulse rate 90 per min., regular, vessel wall not palpable. Blood Pressure:- 180/100 mm. Hg.; Apex beat:- 5th Intercostal space, within the mid-clavicular line. Heart sounds: Closed.
Investigations:- (1) X-Ray of skull:- No abnormality detected, sinuses clear; (2) Urine exam.:- Nil to note; (3) Fundal exam.:- Normal.
Diagnosis:- Menopausal Disturbance.
Treatment:- Phenobarbitone gr. ½ T.i.d. for 3 weeks; (2) Stilboestrol 1 mgm tab. once a day for 3 weeks.
Result:- At the end of 3 weeks:- Symptoms improved, although occasional headache still complained of. Her blood pressure 180/100 mm. Hg and pulse remains the same, i.e. 90 per minute. She was asked to continue stilboestrol 1 mgm. tab. once a day for three months, by which time the patient became symptom free. Headache completely relieved.
Blood pressure reduced to 140/90.

Case 42:- Female, 46, Housewife, married, wt. 12 st.
Complaint:- Since the last two months throbbing frontal headache worse on rising in morning, lasting about 20 minutes, associated with flushing and giddiness, and aggravated by sudden movement, moderate in severity.
Previous Illness:- Nil. Irregular menstruation for the last 2 months. All pregnancies normal.
Family History:- Not relevant.
On examination:- An obese, cheerful woman of average intelligence.
Cardiovascular System:- Pulse rate: 80 per min., regular and vessel wall not palpable. B.P. 190/100 mm. Hg. Apex Beat:- 5th left intercostal, within mid-clavicular line. Heart sounds: closed.
Nil to note in abdomen.
Investigations:- X-Ray of Gall-bladder:- No abnormality detected; X-Ray of stomach after Barium Meal:- Nothing to note; Urine exam.:- Normal; Fundal Exam.: Normal.
Diagnosis:- Menopausal Disturbance.
Treatment:- (1) Mag. Trisilicate i. T.i.d.; (2) 1200 Calories diet; (3) Phenobarbitone gr. ½

T.i.d. for one month.; (4) Stilboestrol 1 mgm tab. once a day for one month. At the end of one month:- only slight improvement. Prescribed Stilboestrol 1 mgm tablet once a day for further periods of 3 months, and she was seen again in six months time. Now flushing was less, headache completely relieved, B.P. and Pulse rate remains the same.

Case 43:- Female, 45, unmarried, weight 10 stones.
Complaint:- Throbbing frontal headache on rising in morning and on straining at stool, lasting half an hour with flushing and sweating. Moderate in severity, Duration - 1½ months.
Previous Illness:- Nil. Menstruation stopped since two months. Family History:- Father died, cause unknown. Mother aged 70, suffering from Hypertension.

On Examination:- Well-built middle-aged woman. Tendency to constipation.
Cardiovascular System:- Pulse rate: 80, regular in time and force. Good volume, vessel wall not palpable. B.P. 150/100 mm. Hg. Apex Beat:- 5th left Intercostal space, within mid-clavicular line. Heart sound - Normal.
Diagnosis:- Menopausal Disturbance.
Treatment :- (1) Diet with roughage to avoid constipation; (2) Phenobarbitone gr. ½ T.i.d. for one month; (3) stilboestrol mgm 1 once a day for one month. Seen after two months:- The patient was much better, B.P. reduced to 120/80 mm. Hg., Headache relieved, flushing less and felt general feeling of well-being. Pulse remains the same.

Case 44:- Female, 47, Housewife, married, wt. 10 st. 2 lbs.
Complaint:- Throbbing frontal headache on rising in morning, lasting ½ an hour, moderate in severity, aggravated by sudden movement of the head and associated with flushing; Duration - 8 weeks.
Previous Illness:- Nil. Irregular periods since 3 months. Family History:- Not relevant.
On examination:- Nervous and flushed female.
Cardiovascular System:- Pulse rate: 70 per min., regular, and vessel wall not palpable. B.P. 160/105 mm. Hg. Apex beat:- 5th left Intercostal space, within Mid-clavicular line. Heart sounds - Normal.
Investigations:- (1) Gynaecological exam.:- No abnormality detected; (2) X-Ray of skull for Sinuses: Negative; (3) Fundal exam.:- Normal; (4) Urine Exam.:- Normal.
Diagnosis:- Menopausal Disturbance.
Treatment:- (1) Phenobarbitone gr. ½ T.i.d. for one month; (2) Stilboestrol I mgm once a day for 1 month.

After one month:- Phenobarbitone stopped, while Stilboestrol 1 mgm. a day continued for 2 months.
At the end of 3 months:- The patient became symptom-free, though B.P. remains the same. Headache relieved and flushing stopped. Periods not completely stopped.
At the end of 6th month:- B.P. 140/90; Pulse rate same, headache relieved and Treatment completely stopped.

Case 45:- Female, 41, Housewife, married, wt. 9 st. 12 lbs.
Complaint:- Throbbing frontal headache worse in morning on sudden rising from bed, with flushing and sweating, lasting ½ hour; Moderate in severity. Duration - 8 weeks.
Previous Illness:- Not relevant. Menstruation was regular but stopped for the last three months; all pregnancies normal.
Family History:- Mother aged 69, suffering from high blood pressure. Four children all alive and well.
On examination:- A nervous woman.
Cardiovascular System:- Pulse rate: 90 per min., regular and vessel wall not palpable. B.P. 160/110 mm. Hg. Apex beat:- 5th left Intercostal space within Mid-clavicular line. Heart sound - Normal. Nil to note in abdomen.
Investigations:- (1) Urine: Normal; (2) Fundal Exm.: Normal.
Diagnosis:- Menopausal Disturbance.
Treatment:- (1) Phenobarbitone gr. ½ T.i.d. for one month; (2) Stilboestrol 1 mgm. once a day for one month.
At the end of one month:- The patient still complained of her symptoms. Headache still present, but blood pressure reduced to 140/90 and pulse reduced to 70 per minute.
Treatment continued and seen after one month; Now symptoms were lessened, headache relieved, but still have slight flushing.
During her the treatment, the patient started her period only for two days at the end of one month's treatment, and now completely stopped. At end of the treatment, the B.P. was 140/90 mm. Hg.

Case 46:- Female, 46, unmarried, wt. 10 st. 2 lbs.
Complaint:- Throbbing frontal headache during the day increased by stooping, lasting ½ an hour with flushing. Moderate in severity; Duration - 2½ months.
Previous Illness:- Nil.
Menstrual History:- Regular till 2 months ago; stopped abruptly.
Family History:- Not relevant.

On Examination:- Flushed and excitable lady.
Cardiovascular System:- Pulse Rate: 100 per min., regular, vessel wall not palpable. B.P. 150/100 mm.Hg. Apex beat:- 5th left Intercostal space within mid-clavicular line. Heart sound closed.
Abdominal examination revealed no abnormality.

Investigations:- (1) Gynaecological exam.:- Normal; (2) Urine - Normal; (3) X-Ray of sinuses - Normal; (4) Fundal exm.:- Normal.
Diagnosis:- Menopausal Disturbance.
Treatment:- (1) Phenobarbitone gr. ½ T.i.d. for one month; (2) Stilboestrol mgm I. one day for one month.
At the end of one month:- B.P. fell to 130/85 mm. Hg. Pulse rate 70 per min: Occasional headache, but flushing much less.
At end of 2 months:- B.P. remained same; headache completely relieved but slight flushing complained of. Did not menstruate during treatment. Treatment discontinued.

Case 47:- Female aged 46, Housewife, married, wt. 11 st.
Complaint:- (1) Attacks of flushing, giddiness and palpitation for the last 3 months; (2) Complained of no headache.
Previous Illness:- Sciatica 4 years ago; Periods stopped a month ago.
Family History:- Father died of gastric carcinoma; Mother died of cancer of uterus; Two children, both alive and well.
On Examination:- Overweight for lady.
Cardiovascular System:- Pulse rate: 90 per min. Regular in time and force; vessel wall not palpable. B.P. 160/100 mm. Hg. Apex beat:- 5th Intercostal space within mid-clavicular line. Heart sounds closed.
On abd. exam.:- No abnormality detected.

Investigations:- (1) Urine - Normal; (2) Fundal Exm.: Normal.
Treatment:- Phenobarbitone gr. ½ T.i.d. for one month; stilboestrol mgm I. once a day for one month.
At end of one month:- B.P. remained same; Flushing and palpitation were less.
At end of 2nd month:- Flushing even less, B.P. 140/90 mm. Hg; Pulse 85 and regular.
Patient was lost sight of and did not report back.

Case 48:- Female, 48, Housewife, married, wt. 9 st.10 lbs.
Complaint:- (1) Breathlessness on exertion for the last 3 months; (2) Throbbing frontal headache worse on rising in morning with flushing and increased by sudden movement of the head. The attack moderate in severity, lasting about ½ an hour. Duration - 3 months.

Previous Illness:- Nil.
Menstruation History:- Periods stopped 6 months ago, started 2 months after that for 2 months and then completely stopped. Pregnancy normal.
Family History:- Father died at 50 of cerebral haemorrhage; Mother aged 76, alive and well; children alive and well.
On examination:- Nervous agitated lady. Pulse rate: 100 per min., regular, and vessel wall not palpable. B.P. 160/110 mm. Hg. Apex beat:- 5th left Intercostal space within mid-clavicular line. Heart Sounds ; 2nd aortic sound accentuated; B.P. 160/110 mm. Hg.
On abd. examination: No abnormality detected.
Investigations:- (1) Urine: Normal; (2) Fundal exam.: Stage I.; (3) E.C.G.: Normal.
Diagnosis:- Menopausal Disturbance.

Treatment:- (1) Phenobarbitone gr. ½ T.i.d. for one month; (2) Stilboestrol mgm. I once a day for one month.
At end of one month: Headache relieved but still complains of flushing. B.P. remains same; Pulse 70 per min.
At end of 3 months: Flushing improved; B.P, remained same. Patient did not report again.

Systolic Hypertension.

Case 49:- Male, aged 50, Paper-worker, Married; wt. 11 st. 9 lbs.
Complaint:- Loss of weight for 1 year; (2) Exophthalmic for 5 months; (3) Nervousness and palpitation for the last 5 months; (4) Fulness in the neck for the last two months; (5) Never had any headache.

A year ago, patient's weight was 12½ stones. Since that time he noticed gradual loss of weight, and becoming increasingly nervous and complained of attacks of palpitation for the last 5 months. Both eyes getting prominent. Had two motions a day but no diarrhoea. He also noticed slight fulness in the neck for the last 2 months. Sweats a good deal, but had good appetite.
Previous Illness:- Influenza during most winters until 3 years ago.
Habit:- Smokes 15 cigarettes a day and 2 pints of beer a month.
Family History:- Wife aged 46, alive and well ; Children ; Nil. Father died of Gastric Carcinoma. Mother died of strangulated Hernia.
General Exam.: A thin anxious-looking man with prominent exophthalmos. Temperature normal on admission. No Cyanosis and no venous engorgement; skin is warm and moist.

Cardiovascular System:- Pulse Rate:- 112 per minute, regular, good volume and arterial wall not palpable.
Blood Pressure: 176/90 on admission mm. Hg.
Apex Beat:- 6th left Intercostal space outside the Mid-clavicular line. Auscultation:- Mitral 1st sound accentuated,
Central Nervous System:- Bilateral exophthalmos, Tremor of hands and all reflexes are present and

Endocrine System:- The thyroid gland is enlarged and easily palpable. Both lobes uniformly enlarged and soft and moves on swallowing.
Investigations:-(1) Urine exm.: Normal; (2) Fundal exm.: Normal; (3)Total white count on admission 6400 per c. m.m.; Total white count on discharge 7000 " " .
Treatment:- (1) Mental and physical bed-rest; (2) High caloric diet; (3) Phenobarbitone gr. i T.i.d. for a week; Phenobarbitone gr ½ B.i.d. for 3 weeks. (4) Methyl Thiouracil 0·2 gm. T.i.d. for 3 weeks, then 0·1 gm a day as a maintenance dose.
Result:- (1) The patient discharged with symptom-free; (2) Pulse Rate dropped to 84. on discharge; (3) Blood Pressure 180/80 mm. Hg; (4) Weight increased to 12 stones 3 lbs; (5) Exophthalmos persisted,

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Case 50:- Male, aged 11; Occupation - School-boy. wt. 5 stone 8 lbs.
Complaint:- Nervousness for 4 years; gradual swelling in the neck for 3 weeks. No headache.
History:- The patient had been nervous for a long time since the age of 7 years. Had to work very hard at school for the last 18 months. During the last three weeks, he noticed the swelling in the neck. The swelling seems less prominent in the morning and more prominent in evening. Also complained of occasional attacks of palpitation. Appetite very good.
Previous Illness:- Measles at the age of 4 years. Mumps at the age of 10 years; Chicken-pox at the age of 6 years,
Family History:- Father, aged 40, alive and well. Mother, aged 32, alive and well. Sisters and Brothers - none.
General Exm.:-(1) A nervous little boy, with obvious swelling in the neck and slight exophthalmos. The swelling moves on swallowing. Skin moist and sweating.

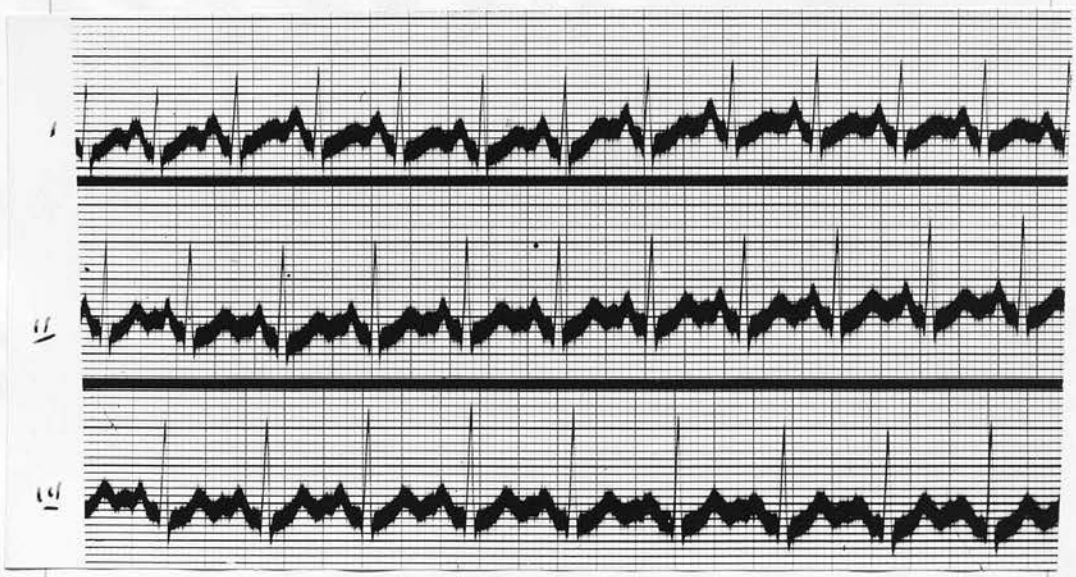
Cardiovascular System:- Pulse rate 120 per min and regular. B.P. 150/60 mm. Hg. on admission.
Apex Beat:- 5th left Intercostal space within the mid-clavicular line. Heart sounds - Normal.

Cn.

Cardiovascular System:- Fine tremors of hands, all reflexes are present and equal.
Endocrine System:- The thyroid is diffusely enlarged. Systolic murmur heard over the gland, and gland moves on swallowing.
Special Investigations:- (1) B.M.R. + 30%; (2) Blood cholesterol 154 mgm%; (3) E.C.G.:- Sinus tachycardia; (4) Total W.B.C. on admission 8000 per c. mm., Total W.B.C. on Discharge 8000 per c. mm.; (5) Urine Exm.: Normal; (6) Fundal Exm.:- Normal.
Diagnosis:- Thyrotoxicosis.
Treatment:- (1) Physical and Mental rest in bed; (2) Diet:- High caloric diet; (3) Drugs:- Luminal gr. i. B.i.d. for 1 week, then gr. ½ a day; thiourocil 0·2 gm. B.i.d. for 3 weeks, then 0·1 Gm. - one dose on alternate days after 3 weeks.
Result:- (1) Patient discharged with symptom free; (2) Pulse relieved to 84 per min. regular; (3) B.P. fell to 120/70 mm. Hg; (4) Weight increased to 6 st. 2 lbs.; (5) Slight Exophthalmos.

Case 51:- Female, 31, typist, single, wt. 7 stone.
Complaint:- (1) Exophthalmos for 3 weeks; (2) Weakness and lethargy since 3 weeks.
History:- The patient developed a sore throat a month ago. Since then she became nervous and had some palpitation and sweating frequently and profusely at times.
Previous Illness:- Measles at 4 years of age; Menstrual History: Normal. No headache.
Family History:- Father a ged 60, alive and well ; sisters, aged 28 and 25 - both alive and well. Mother died 13 years ago - Pulmonary Tuberculosis.
General Exm.:- Apprehensive, anxious-looking with prominent eyes. Skin is moist, warm and sweating.
Cardiovascular System:- Pulse rate 126 per minute, regular; and arterial wall not palpable; B.P. 150/65 mm. Hg.; Apex Beat:- 5th intercostal space, within mid-clavicular line;
Auscultation:- Faint systolic murmur at the mitral Pulmonary area. Endocrine System:- Isthmus and lobes of thyroid palpable and moves on swallowing.
Central Nervous System:- Prominent exophthalmos and reflexes present and equal.
Special Investigations:- (1) Blood Cholestrol - not done; (2) B.M.R. + 31 %.; (3) Total W.B.C. per c. mm. before ; (4) Total W.B.C. 6800 per c. mm. on discharge; (5) Urine:- Normal; (6) Fundal Exm.:- Normal; (7) E.C.G. - not taken.
Diagnosis:- Hypothyroidism.
Treatment:- (1) Physical and mental rest in bed; (2) High caloric diet; (3) Luminal gr. i. T.i.d. for 1 week; Luminal gr. ½ B.i.d. for 3 weeks; Methyl Thiourocil 0·2 gm. T.i.d. for 3 weeks, then 0.2 gm a day.
Result:- Patient discharged symptom free; (1) Pulse rate dropped to 80 per min., Regular; (2) B.P. fell to 120/70 mm. Hg,; (3) wt. rose to 8 st.; (4) Exophthalmos unaffected.

CASE Nº 52



Case 52:- Female, 23, single, wt. 10 st., 1 lbs.
Occupation:- Domestic servant.
Complaint:- Swelling in the neck for 5 years; Sweating for 5 years; Breathlessness on exertion for 1 year; Frontal headache for 6 months.
History:- For the last 6 months, complained of throbbing frontal headache on exertion, lasting a few minutes and relieved by rest. Periods irregular since one year.
Previous Illness:- Measles during childhood;
Habit:- 45 cigarettes a day.
Family History:-Father aged 70 alive and well; Mother died of cerebral haemorrhage at 60 years; Two brothers and two sisters all alive and well.
General Exm.:-A well-built girl, with staring eyes and fulness in the neck; fine tremors. Sweating and soft moist skin.
Cardiovascular System:- Pulse rate 110 per min., Regular and vessel wall not palpable.
B.P. 160/60 mm. Hg.; Apex Beat:- 5th Intercostal space ½ inch outside the Mid-clavicular Line.
Auscultation:- Systolic Murmur at the mitral and pulmonary area.
Endocrine System:- Thyroid gland uniformly enlarged with systolic murmur audible over it.
C.N. System:- Fine tremors, all reflexes are present and equal.
Special Investigations:- (1) B.M.R. + 40%; W.C. 6000 on admission; on discharge 7000; (2) E.C.G. Sinus tachycardia; (3) X-Ray:- Slight compression of the trachea, but no difficulty in swallowing; (4) Blood cholestrol not done; (5) Urine exm.: Normal ; (6) Fundal Exm.: Normal.
Diagnosis:- Hypothyroidism.
Treatment:- Mental and Physical rest in bed; (2) High caloric diet; (3) Phenobarbitone gr. o. T.i.d. for 1 week; gr. ½ B.i.d. until her discharge, Thiouracil/methyl Methyl Thiouracil 0.2 gm. T.i.d. for 3 weeks; 0.2 gm. a day as a maintenance dose.
Result:-(1) The patient discharged with symptom free; (2) Headache much relieved; (3) Pulse dropped to 80; (4) B.P. dropped to 120/60 mm. Hg.; (5) Weight increased to 11 st. 8 lbs.

Case 53:- Female, 45, Housewife, married, wt. 7 st. 8 lbs.
Complaint:- Loss of weight for 4 months; Tremors of hands for 14 days.
Two years ago her husband had an attack of Coronary Thrombosis and recovered from the attack. Since that time, the patient had more work to do. Four months ago, she noticed the losing of weight, and getting increasingly nervous and sweating a great deal. No headache.

Previous Illness:- Prolapsed Uterus treated surgically in 1938; Appendicectomy 1939; Pregnancy normal.
Family History:- Father died at 73, cerebral haemorrhage; mother died at 52, Appendicitis; Children two, aged 20 and 13; both well.
General Exm.:- Anxious-looking woman with slight exophthalmus.
Cardiovascular System:- Pulse rate 110 per minute. Regular in time and force. B.P. 150/70 mm. Hg. On discharge 130/70. Apex.Beat:- 5th left Intercostal space inside mid-clavicular line.
Heart sounds - closed.
Endocrine System:- Thyroid gland diffusely enlarged and systolic murmur heard on auscultation.
C.N. System:- Tremors of hands and all reflexes are exaggerated.
Special Investigations:- B.M.R. + 38%.; W.B.C. on admission 8000 per c. mm.; W.B.C. on discharge 6800 per c. mm.; Urine Exm.:- Normal; Fundal Exm.:- Normal.
Diagnosis:- Hyperthyroidism.
Result:- (1) The patient discharged with symptom free; (2) Pulse 82; (2) B.P. 130/70; (3) Wt. increased to 8 st. 6 lbs.; (4) Slight exophthalmos persisted.
Treatment:- (1) Mental and physical rest.
(2) High caloric diet; (3) Luminal gr. i. T.i.d. for a week, reduced to gr. ½ B.i.d.; (4) Methylthiouracil 0.2 gm. T.i.d. for 3 weeks, then 0.2 gm. a day as a maintenance dose.

Case 54:- Female, 19, Housewife, married; wt. 8 st.8 lbs.
Complaint:- Slight swelling in neck for 1 year; Prominent eyes for 1 month; Breathlessness on exertion for 1 month.
History:- About a year ago, the patient noticed a slight swelling in the neck; 4 months ago, her mother died from cerebral haemorrhage, since then she became increasingly nervous and swelling in neck increased in size. Her eyes being slightly prominent and complains of breathlessness on exertion for the last month. No headache.
Previous Illness:- Scarlet fever at the age of 6 years.
Family History:- Mother died of cerebral haemorrhage aged 56; father suffers from Rheumatoid arthritis.
General Exm.:- A nervous woman with exophthalmus.
Cardiovascular System: Pulse 100 per min. and regular. B.P. 150/60 mm. Hg. Apex Beat:- Within normal limit; Heart sound normal.
Endocrine System:- Smooth diffuse enlargement of both lobes of thyroid.
C.N. System:- Tremors of hands and reflexes are
Special Investigations:- (1) Total W.B.C. 5200 per c. mm on admission; 6000 per c. mm. on discharge; Fundal and Urine:- Normal.
Treatment:- (1) Mental and physical rest; (2) High caloric diet; (3) Luminal gr. T.i.d. for a week,then

gr. ½ B.i.d.; (3) Methyl Thiouracil 0.2 gm. T.i.d. for 3 weeks, and 0.2 gm. as a maintenance dose.

Case 55:- Female, 55, clerkess, unmarried, wt. 7 st. 6 lbs.
Complaint:- Throbbing headache for 6 months; loss of weight for 9 months; Nervousness and sweating for 6 months; Exophthalmus.
History:- Her friends noticed that her eyes had been getting bigger for the last 6 months; she also complained of breathlessness on exertion. Sweats a good deal; attacks of palpitation and occasional attacks of diarrhoea. Appetite excellent. For the last six months, the patient complained of throbbing headache situated behind the eyes, worse on reading, lasting for 10, minutes and relieved by rest.
Menopause at the age of 48. Habit:- Nil.
Previous Illness:- Scarlet fever 1912; Herpes Zoster 1943.
General Exm.:- Thin, excitable,breathless woman with marked exophthalmus. Pulse Rate:- 110 per min, regular; B.P. 200/110 mm. Hg.; Apex Beat: 5th Space just outside Mid-clavicular line. Auscultation: Systolic murmur at the mitral area.
Endocrine system:- Thyroid gland enlarged uniformly and systolic murmur heard over it.
Special Investigations:- B.M.R. + 34%.; fell to + 15% after treatment; E.C.G. Sinus tachycardia; Total W.B.C. 7200 per cmm on admission; 7100 per cmm. on discharge.
Diagnosis: Hyperthyroidism.
Treatment:- (1) Mental and physical rest; (2) Usual thiouracil therapy had no effect. Therefore she was treated as follows:- Stilboestrol 5 mgm a day for 6 weeks; Deep XRay - 10 exposures.
Result: Patient improved; Exophthalmos persisted; Pulse rate 70 per min on discharge. Headache relieved.

Case 56:- Female, 43, Housewife, married, wt. 7 st. 13 lbs.
Complaint:- Nervousness for 16 months; Frontal headache for 10 months; Swelling in neck and loss of weight for 6 months. The patient complained of nervousness and attacks of palpitation and excitement during the last 16 months, felt tired and listless and sweats a good deal. Troubled with throbbing frontal headache on exertion, lasting about 10 minutes and relieved by rest. Duration - 10 months.
No Exophthalmos.
Previous illness:- Diphtheria at the age of 16 yrs.
Menstrual History - Normal.
Family History:- Husband aged 45, alive and well.
Children - none. Father died of cerebral haemorrhage 73; mother died of gastric cancer, 75.
General Exm.:- Well-nourished middle-aged woman, with rather flushed face and very nervous and tremors of hands.

C.V. System:- Mitral wall not palpable.
Pulse: 100, regular; B.P. 150/70 mm. Hg. on admission; Apex Beat: Within normal limit. Heart sounds - Normal. Thyroid moderately enlarged, firm and smooth; Systolic murmur heard on auscultation. C.N. System: Tremors of hands, all reflexes are
Special Investigations:- B.M.R. + 27 ;
E.C.G. Sinus Tachycardia;
Total W.B.C. 5000 per cmm. on admission, 6000 per c.mm. on discharge.
Diagnosis: Thyrotoxicosis.
Treatment:- (1) Mental and physical rest;
(2) High caloric diet; (3) Phenobarbitone gr. i. T.i.d. for 1 week, then ½ B.i.d. for 14 days.
Methyl Thiouracil 0.2 gm. T.i.d. for 3 weeks, thereafter 0.1 gm. a day as maintenance dose.
Result:- (1) Discharged as Symptom-free; (2) B.P. 130/70; (3) Pulse 80; (4) Wt. increased to 8 st.8 lbs; (5) Headache relieved.

Case 57:- Female, 21; waitress, unmarried - wt. 10 st. 2 lbs.
Complaint:- Nervousness for 1 year; Exophthalmos for 6 months; Breathless on exertion for 4 months. Difficulty in swallowing for 1 month. Frontal headache for 1 month.

A year ago her mother died, ever since, she has been very nervous, extremely irritable and easily frightened. She has been breathless on exertion for the last 3 - 4 months and had attacks of palpitation. Sweats a good deal, and noticed that her eyes were getting prominent for the last 6 months. There has been some difficulty in swallowing food for the last month. She had frequent frontal headache with occasional blurring of vision, for the last month. This frontal and throbbing headache lasts for 5 minutes, worse on excitement and relieved by rest.
Previous Illness: Pleurisy at the age of 11 years.
Habit:- Nil. Family History: Not relevant.
On examination:- A young girl, very restless, flushed face, prominent eyes, skin warm moist and soft, with sweating palms.
C.V. System:- Pulse: 110 per min. and regular. B.P. 160/70 mm. Hg on admission; Apex Beat; Within mid-clavicular line. Systolic murmur at the Pulmonary area. Endocrine System: Uniformly enlarged thyroid; Systolic murmur heard over the gland.
Special Investigations: B.M.R. +40%. E.C.G. Sinus tachycardia.
On admission W.B.C. 4800 per c.mm. on discharge W.B.C. 6000 per c. mm.
Diagnosis:- Hyperthyroidism.

Treatment: Rest. In view of the retro-sternal goitre she was prepared for operation by Iodine therapy. Phenobarbitone gr. i. T.i.d. for 10 days; Lugol's iodine 5 m T.i.d. for 10 days, then she was operated upon. Subtotal thyroidectomy and made uneventful recovery.
Result:- Discharged symptom-free. (1) Pulse dropped to 80 per min. and regular; (2) B.P. 130/70 mm.Hg. (3) Weight 11 st. 2 lbs.; (4) Exophthalmos - persisted.

Case 58:- Female, 24, domestic servant, unmarried. wt. 7 st. 12 lbs.
Complaint:- Nervousness for 2 months. Two months ago she noticed that she was becoming nervous and developing fulness in the neck, with staring eyes. She had attacks of palpitation. No headache.
Previous Illness:- Nill. Periods regular.
Family History - Not relevant.
General exm.:- Excitable girl with exophthalmos, skin moist and warm and has tremor of the hands.
C.V. System:- Pulse rate: 130 per min and regular; B.P. 190/80 mm. Hg on admission. Apex Beat:- forcible and within Mid-clavicular line. Heart sound - Normal.
Endocrine System:- Thyroid gland uniformly enlarged, firm and smooth. Systolic murmur audible over the gland.
C.N. System:- All reflexes are brisk and equal.
Special Investigations:- B.M.R. +15%.
W.B.C. 3000 per c.mm. on admission; W.B.C. 4000 per c.mm. on discharge.
Treatment:- (1) Mental and physical rest; (2) High caloric diet; (3) Luminal gr. i. T.i.d. for 1 week, then Luminal gr. ½ B.i.d.; (4) Methyl thiouracil 0.2 gm. T.i.d. for 3 weeks, then 0.1 gm. a day as a maintenance dose.
Result:- Symptom free; Exophthalmos persisted. B.P. 130/70 mm. Hg. on discharge. Pulse 80 per minute and regular.

Cases of Aortic Incompetence.

Case 59:- Female, 58, married, housewife; wt. 11 st.6 lbs.
Complaint:- Breathless on exertion for 2 years; Throbbing Frontal headache - 2-3 months; Anginal pain on exertion - 2 months.
History:- Two years ago the patient began to suffer from dyspnoea on exertion. Dyspnoea gradually increased and recently has been occurring at night as well. For the last two months there has been Anginal pain over the precordium and along the left arm, on

exertion, also complains of attacks of dizziness which followed by throbbing frontal headache, lasting for 15 minutes and relieved by rest.
Previous Illness: Nil. Habit:- Nil.
Menopause at the age of 47 years.
Family History:- Husband died of heart trouble two years ago; Father died at the age of 64 - Cause unknown. Mother died at the age of 70 from Pneumonia; Children - none.
General Exam.:- stout pale female, propped up in bed; visible pulsations in the neck. Temp. normal.
Pulse Rate: 110 on admission, "water hammer" - collapsing - arterial wall not palpable.
B.P. 200/70 mm. Hg. on admission. Heart: Apex Beat: 6th Intercostal space in Ant. axillary line.
Auscultation: Aortic systolic and aortic diastolic murmur. The diastolic murmur conducted down the left border of the sternum.
Respiratory:- Respiration 25.
Lungs:- Bilateral basal crepitation.
Alimentary system: Liver and spleen not palpable.
Special Investigations:- X-Ray of Heart: Enlarged left ventricle, and basal crepitation; E.C.G. Left axis deviation; Fundal Exm.: Normal. Urine Exm.:- 700 c.c. output on admission. sp.gr. 1020.
On discharge urine output 1200 c.c. with sp.gr. 1015.
No albumin and no sugar.
Wasserman reaction positive of blood and negative of cerebro spinal fluid.
Diagnosis: Syphilitic Aortitis.
Treat:- (1) Rest in bed; (2) Aminophylline 0.2. gm. T.i.d. orally for 10 days; (3) Salt-restricted diet.
Result:- (1) Patient improved symptomatically; (2) B.P. remains the same 200/70 mm. Hg; (3) Dyspnoea relieved; (4) Pulse 80 per min. and regular; (5) Headache relieved.

Case 60:- Male, aged 50, schoolmaster, married, wt. 8st. 13 lbs.
Complaint:- Breathlessness on exertion for 4 yrs. Breathlessness at night for 1 week.
History:- Cough at night for 1 week. The patient had known that he had a bad heart at the age of 10 years, but remained perfectly fit till four years ago, when he began to be progressively breathless on exertion. For the last week he has been breathless at night. No oedema and no headache. Unproductive cough at nights for the last week.
Previous Illness:- Growing pains at the age of 5.
Habit :- No drink and no smoking for the last 4 yrs.
Family History:- Not relevant.
General Exm.:- Temp. normal. No peripheral oedema.
Pulse rate: 110 per min. Irregular in time and force. Wall not palpable. B.P. 190/80 mm. Hg. on admission.
Apex Beat:- 6th Intercostal space outside mid-clavicular line.

Auscultation:- Mitral systolic murmur and rough mid-diastolic murmur at mitral area. Systolic and diastolic murmur at the aortic area, systolic murmur conducted towards the neck. Aortic diastolic conducted down the sternum. Respiratory rate 26 per minute.
Lungs:- Bilateral basal crepitation. Liver and spleen not palpable.
Investigations:- E.C.G.: Left axis deviation and auricular fibrillation; (2) XRay: Left ventricular enlargement and bilateral basal crepitation.
(3) Wassermann Reaction - Negative; (4) Fundal Exm.:- Normal; (5) Urine Exm.:-
On admission:- 500 c.c. Output in 24 hrs. sp.gr. 1020 and trace of albumin; on discharge:- output 1600 c.c. with sp.gr. 1015.
Diagnosis:- Rheumatic Valvular Disease of the heart.
Treatment: Rest; (2) Diet - Salt restricted diet; (3) Digoxin 0.5 mgm T.i.d. for 3 days orally, thereafter 0.25 mgm B.i.d. as a maintenance diet, also Aminophylline 0.2 gm. T.i.d. for one week, then Mersalyl one injection 2 c.c. 1·m·l - resulted in profuse diuresis.
Result:- (1) Discharged as Rheumatic Heart Disease - improved; (2) B.P. remained the same 190/80 mm. Hg; (3) Pulse Rate dropped to 70 per min, but irregular.

Case 61:- Male, 57, Married, wt. 10 st. 6 lbs.
Occupation: Iron-Moulder.
Complaint:- Breathlessness on exertion for 18 mths. Tightness in chest on exertion for 18 months ; Swelling of ankles at night for 1 month; Breathlessness at night for 1 month.
Eighteen months ago the patient began to have breathlessness on exertion, associated with feeling of constriction in the chest. This ceased when effort stopped. During the last month, had swelling of both ankles at night and has been breathless when in bed. No headache.
Previous Illness: Severe burn 14 years ago. No history of Rheumatic fever.
Habit:- Smokes and drinks moderately.
Family History:- Nil to note.
On examination: A red-faced man, propped up in bed; oedema of both ankles and slight congestion in vessels of the neck. Pulse Rate: 80 per min., regular. collapsing wall slightly thickened. B.P. 175/75 mm. Hg. on admission; Apex Beat: 6 inches from mid-sternal line in 6th left intercostal space.
Asucultation:- Aortic Systolic and diastolic murmur. The latter conducted down the sternum.
Respiration Rate:- 25 per min.
Lungs:- Bilateral Basal crepitation. Liver and spleen not palpable.
Investigations:- E.C.G. Left axis deviation.
X-Ray :- marked enlargement of left ventricle and basal crepitation and calcified aortic cusps.

W.R. Negative. Fundal Exm.: Normal.
Urine:- Initial output on admission 1 litre; sp.gr. 1018 and trace of albumin, thereafter output 1500 c.c. sp.gr. 1015. No albumin and no sugar.
Diagnosis:- Calcification of Aortic Cusps.
Treatment:- (1) Rest; (2) Low-salt diet; (3) Pul Digitalis grs. T.i.d. for 1 week, then Pul digitalis gr. 1½ once a day.
Result:- (1) Improved on discharge; (2) Weight fallen to 10 st. 4 lbs; (3) B.P. Remains the same 115/75 mm. Hg.; (4) Pulse dropped to 70 - Regular.

Case 63:- Female, aged 46, housework, married, wt. 12 st. 8 lbs
Complaint:- Admitted with Acute Gastro-Enteritis. Never had any headache and never had any cardiovascular system. But she told that she had had bad heart following Rheumatic fever.
Previous Illness:- Rheumatic Fever at the age of 15 years. No headache.
Habit :- Nil. Menstruation - Normal.
Family History:- Nil to note.
General Exm.:- A well-built woman with slight malar flush. Temperature:- 99·8°F.
C.V. System:- 100 per min. Irregular in time and force -
B.P. 160/70 mm. Hg. on admission. Apex Beat :- 5th space outside Mid-clavicular Line.
Auscultation:- (1) Systolic and mid-diastolic murmur at mitral area; (2) Systolic and diastolic murmur at the aortic area; diastolic murmur conducted down the sternum.
Investigations:- E.C.G. Auricular fibrillation; Fundal Exm.: Normal; W.R. - Negative.
Urine:- Initial output 800, sp.gr. up to 1022; on discharge output 1500 and sp.gr. 1015- no albumin and no sugar.
Diagnosis:- Acute Gastro-Enteritis and Rheumatic Valvular Disease.
Treatment:- (1) Rest; (2) Dehydration treated with fluids; The gastro-enteritis settled down, and later given normal diet.
Result:- (1) The Patient recovered from Gastro-Enteritis and discharged; (2) B.P. remained the same - 160/70 mm. Hg; (3) Pulse rate 75 on discharge - Irregular.

Case 63:- Male, aged 51, married, Miner, wt. 10 st. 7 lbs.
Complaint:- Severe Breathlessness for 1 week.
History:- Severe breathlessness for a week before admission to the Hospital. The patient was quite well until a week ago. Suddenly complained of breathlessness while lifting a heavy weight. Since then breathless on slight exertion. Slight frontal headache on exertion for the last six months and relieved by rest.
Previous Illness:- Pleurisy 30 years ago.
Habit:- 10 cigarettes a day. Family History - Nothing to note.
General Exm:- Healthy-looking man with breathlessness. No visible pulsation in the neck. Pulse Rate:- 90 per min., regular. B.P. 200/90 mm. Hg. on admission.
Apex-Beat:- 5th space outside Mid-clavicular line.
Auscultation:- Diastolic murmur at aortic area, conducted down along the left border of sternum. Pulmonary 2nd sound accentuated.
Respiration: 26 per minute. Bi-lateral Basal crepitation. Liver and spleen not palpable.
Investigation:- E.C.G. Left axis deviation.
X-Ray:- Left Ventricular Hypertrophy and fine reticulation in the lungs. early Silicosis.
W.R. - Positive. Fundal exam.:- - Normal.
Urine Exam.:- Initial output on admission 1000 c.c. sp.gr. 1022. No albumin. On discharge:- Output 1700 c.c and sp.gr. 1017: No albumin.
Diagnosis:- Syphilitic Aortitis with Incompetence.
Treatment: (1) Rest; (2) Amenophylline 0.3 gm. T.i.d. orally for 14 days, then transferred to Venereal department.
Result:- Left ventricular improved. Dyspnoea relieved; Headache relieved. B.P. Remains same 200/90 mm. Hg. Pulse 74 per min. and regular.

Case 64:- Male, 30, Linen-factory-worker, married wt. 10 st. 10 lbs.
Complaint:- (1) Sudden pain in right arm for 4 days; (2) Throbbing frontal headache on exertion since 6 months.
History:- The patient has been perfectly fit since 4 days ago, felt sudden pain in right arm, which became numb and useless. Frontal throbbing headache on exertion lasting few minutes and relieved by rest. Duration - 6 months. Habit:- Moderate.
Previous Illness:- Rheumatic fever at the age of 7 years and again at the age of 14 years.
On Examination:- A healthy-looking man with right arm completely white; visible pulsation in neck.
Cardiovascular System:- Pulse rate: 82 per min., Irregular. B.P. 160/60 mm. hg on admission.
Apex beat:- 6th interspace outside mid-clavicular line.
Auscultation:- Mitral mid-diastolic murmur and aortic diastolic murmur conducted down the sternum

Respiration Rate : 24 per minute. Lungs:- No basal crepitation. Liver and spleen not palpable.
Right arm:- cold and pulseless.
No investigation done as patient did not live long enough.
Diagnosis:- Embolism in right Brachial Artery with Rheumatic Valvular Disease of the Heart.
Treatment:- (1) Rest; (2) Heparin 10,000 l.v.l. thereafter 6000 units 6 hourly l.v.i.
The Patient developed cerebral embolism and suddenly died.
Post mortem Findings:- Rheumatic Mitral stenosis and Rheumatic Aortic Incompetence; Embolism in rt. Brachial Artery and in Left Internal carotid artery; Intramural clot in left auricle.

Case 65:- Female, 25, Unmarried; wt. 10 stones.
Complaint:- Pains in wrists and knees for a week.
History:- The patient was perfectly fit till a week ago when she developed pains in joints and began to sweat a good deal and felt ill. The pain was fleeting in character. No headache.
Previous Illness:- Rheumatic fever at 14, and again at 20. Habit - Nil. Menstruation - regular.
Family History - Nil to note.
On examination:- Slightly breathless woman, sweaty and febrile. Temp. 104 on admission.
C.V. System:- Pulse 108 per min, regular, "water hammer"; B.P. 160/20 mm. Hg. Apex Beat:- 6th Space outside mid-clavicular line.
Auscultation - Aortic diastolic murmur conducted down the sternum.
Lungs - Normal.
Joints: Red, swollen and painful knee joints and wrist joints.
Investigations:- E.C.G. P-R interval 0.24 sec.
Urine:- 720 c.c. initial output sp.gr. 1020, no albumin; on discharge 1800 output sp.gr. 1016, albumin nil.
Diagnosis:- Rheumatic Fever with Moycarditis and Aortic Incompetence.
Treatment:- (1) Bed rest; (2) Soda Salicyl gr. 200) a day f
soda Bicar grs. 200) one mon
then sent to Convalescent Home.
Result:- Fever subsided, temp. 97.4; Pulse rate: 88. B.P. 140/20 mm. Hg.

/63
Case 66. Female,/housewife, married,
Complaint:- Dyspnoea for 5 days.
History:- A week ago the patient suffered from Bronchitis, and became, recently, breathless. No headache.
Previous Illness:- Nothing to note.
Family History:- Husband suddenly died due to heart failure at the age of 64 years. Children 2, one stillborn; one child died at the age of one year.
Menopause at 46.

On examination:- A very dyspnoic woman with visible pulsation in neck.
Cardiovascular System:- Pulse Rate:- 100 per min., regular, collapsing vessel-wall not palpable.
B.P. 140/30 mm. Hg. on admission. Apex Beat:- 6th space outside midclavicular line.
Auscultation:- Aortic diastolic murmur conducted over the sternum.
Respiration: 30 per min.
Lungs:- Bilateral coarse crepitation; Liver and spleen not palpable.
Investigation:- not done as patient died.
Diagnosis:- Aortic Incompetence with Acute left Ventricular Failure.
Treatment: (1) Rest; (2) Digoxin 0.5 mgm. l.v.l.
(3) Aminophylline 0.2 gm. l.V.l. - one dose;
(4) Oxygen therapy.
Patient died of Acute Left Ventricular failure.
Post Mortem Findings:- Syphilitic Aortic Incompetence and oedematous. Lungs and left Ventricular Hypertrophy.

Case:- 67 Male, 36, clerk, married; wt. 10 st. 2½ lbs.
Complaint:- Admitted to the ward for an attack of Acute Appendicitis, which was surgically removed. Three days after the operation, the patient became rather breathless at rest in bed.
History:- For the last 3 years the patient noticed slight increase of dyspnoea on exertion. No history of headache.
Illness:- Rheumatic Fever at the age of 16 years.
Habit:- 10+15 cigarettes a day, occasional glass of beer. Family History:- Father died at 58 from Coronary Thrombosis; Mother died at 60 years from Mesenteric Thrombosis.
On Examination: A healthy-looking man with visible pulsation in the vessels of the neck.
Cardiovascular System:- Pulse rate: 100, regular in time and force. "water-hammer". B.P. 200/60 mm. Hg. on admission. Apex Beat:- 7th Intercostal space in Anterior axillary line.
Auscultation:- Systolic and Diastolic murmur at the aortic area, diastolic murmur conducted down the left border of the sternum.
Respiratory Rate:- 28 per min. Lungs:- Bilateral Basal Crepitation. Alimentary: Liver and spleen not palpable; surgical scar in right iliac fossa.
Investigations:- X-Ray of heart:- Left Ventricular Hypertrophy and basal crepitations in Lungs; (2) E.C.G. Left axis deviation; (3) Urine:- Initial output 960 c.c. sp.gr. 1020; later initial output 1500 sp.gr. 1015; albumin and sugar absent; (4) Fundal Exm.:- Normal; (4) Wassermann Reaction - Not done.
Diagnosis:- Rheumatic Aortic Incompetence with Acute Left Ventricular Failure.
Treatment:- (1) Rest; (2) Aminophylline 0.2 gm. T.i.d. orally for 1 week, then 1 Tablet (0.2 gm.) at night for

a further week..
Result:- (1) Discharged with Symptom-free; (2) B.P. remains same 200/60 mm. Hg; (3) Pulse Rate dropped to 80 - regular.

Case 68:- Male, 28, Unmarried, Merchant, wt. 10 st. 2 lbs.
Complaint:- Dyspnoea on exertion for 15 years. Swelling of ankles for 1 month.
History:- The patient complained of Breathlessness on exertion during the last 15 years, Until now, he can only walk about 100 yards; swelling of ankles in the evening during the last month. No Headache.
Previous Illness:- Growing Pains in childhood.
Family History:- Not available. Habit:- Nil.
On examination:- A well-built man with slight dyspnoic.
C.V. System:- Pulse rate:- 110 per min., irregular, "water-hammer" and wall not palpable.
B.P. 180/80 mm. Hg. Apex Beat:- 6th Interspace outside Mid-clavicular line.
Auscultation:- Mid-diastolic murmur at mitral area and aortic systolic and aortic diastolic at the aortic area. Aortic diastolic conducted down the left border of the sternum. Respiration - 24 per min. Liver and spleen not palpable.
Investigations:- (1) X-Ray:- Mitralization of the heart with enlarged left ventricle and pulmonary congestion; (2) E.C.G. Left axis deviation and auricular fibrillation; (3) Fundal Exm.:- Normal. (4) Urine Exm.: 800 c.c. sp.gr. 1024.
Diagnosis:- Rheumatic Valvular Disease of the Heart.
Treatment:- (1) Rest; (2) Digoxin 0.25 mgm. twice a day orally. Three days after admission developed massive Pulmonary infarcts and the patient died suddenly.
Post-Mortem Findings:- Rheumatic Endocarditis - Mitral Stenosis, Aortic-Incompetence and Pulmonary Infarcts.

Case 69:- Male 65, married, Black-smith, wt. 10 st. 6 lbs.
Complaint:- Increasing breathlessness on exertion for 10 years.
History:- Ten years ago, the patient suffered from an attack of Acute Lobar Pneumonia and ever since he complains of increasing breathlessness on exertion. During every winter had severe attacks of "cold" and "coughs" with muco-purulent sputum.
No history of Headache.
Previous Illness:- Acute Lobar Pneumonia 10 yrs. ago.
Habit:- Smokes and drinks moderately.
Family History:- Father died at the age of 58 from Pneumonia; Mother died at the age of 74 from Pneumonia; Children - None.

General Exm.:- An elderly heavily-built man with slight breathlessness, but not in distress.
Cardiovascular System:- No visible pulsation in neck. Pulse rate: 90 per minute, regular in time and force; the arterial wall tortuous, calcified and palpable. B.P. 175/80 mm. Hg. on admission.
Inspection:- The chest is barrel-shaped and Emphysematous. Apex Beat:- 5th left space in Mid-clavicular line.
Ausculation:- Heart sounds - normal at all areas except 2nd sound at the aortic area greatly accentuated.
Respiratory System:- Respiration 24 per min.
Other systems:- No abnormality found.
Investigations:- (1) X-Ray:- Bilateral basal bronchiectasis with Emphysema. Heart-shadow: normal and no unfolding of aorta seen; (2) E.C.G.: Left Axis deviation; (3) Fundal Exm.: Grade I. (4) Urine exm.:- Output 1500 c.c.; sp.gr. 1018. Albumin - Nil; sugar - Nil.
Diagnosis:- Bilateral Bronchiectasis, Emphysema and atheroma of the Aorta.
Treatment:- (1) Bed rest; (2) Breathing exercises- (3) Postural drainage; (4) Syrup Codeine Phosp i. at bed time.
Result:- The patient improved and became symptom free on discharge. B.P. remains same 175/80 mm. Hg. Pulse Rate: 70 per min. Regular.

Case 70:- Male, 61, Unmarried, Miner, wt. 10 st. 10.
Complaint:- Nocturnal Dyspnoea and swelling of legs for a month.
History:- For the last year, the patient has wakened up at night feeling very breathless. Had to sit up in bed, and the attacks gradually wear off. A month ago he noticed the swelling of his legs in the evening and at the same time breathlessness became worse. The patient has had productive cough with green muco-purulent sputum of many years standing.
No History of Headache.
Previous Illness: Malaria and Dysentery 1919.
Habit :- Smokes and drinks moderately.
Family History:- Uneventful.
General Exm.:- A well-built man lying propped up in bed; temperature normal; oedema of sacrum and both legs swollen.
Cardiovascular System:- Pulse rate 100 per min, regular, good arterial wall palpable. B.P. 190/90 mm. Hg. on admission; on Discharge 190/90 mm. Hg.; Apex beat: 6th left Intercostal space, outside mid-clavicular line.
Auscultation:- Heart sounds normal at all areas except 2nd Aortic accentuated.
Respiratory System:- Respiration 24 per minute. Bilateral Basal Crepitations.

Abdominal Exam.:- Lines palpable, 2 finger-breadth below the right costal margin. No clinical Ascites found.
Investigations:- (1) Radiology of Heart:- Enlargement of left ventricle; Unfolding and Calcification of aorta. Early silicosis in both lungsfields; (2) E.C.G. Left Axis deviation; (3) Fundal Exam.:- Grade I.
Urine Examination:- On admission 1000 c.c. output sp.gr. 1020 ; albumin - trace present.
On discharge 1600 c.c. output sp.gr. 1020; albumin - Nil.
Diagnosis :- Atheroma of the Aorta.
Treatment:- (1) Bed rest; (2) Digoxin 0.5 mgm. 4 times a day for 3 days orally. Then Maintenance dose 0.25 mgm. B.i.d. Mersalyl 2 c.c. l.m.l. twice a week for 3 weeks.
Result:- The patient discharged symptom-free. Oedema disappeared; weight fallen to 10 st. 4 lbs. Pulse rate - 80 per min. B.P. Remains the same.

Case 71:- Female, 69, Married, Housewife, wt. 9 st. 2 lbs.
Complaint:- Continuous pain in the chest and in the left arm for 4 hours.
History:- On walking home one night the patient experienced an agonising pain in the chest and in left arm; sweated and felt giddy, No History of Headache.
Previous Illness:- Operated on for Prolapsed Uterus 11 years ago; had diabetes for the last 4 years controlled by dieting only.
Family History:- Husband 72 years alive and well; Children: Three, all alive and well. Parents : Died - cause unknown.
General Exam:- A pale slightly-built lady obviously shocked.
Cardiovascular System:- Pulse Rate: 100 per min. regular in time and force, poor in volume. Arterial wall palpable; on Discharge 68.
B.P.: 120/70 mm. Hg. on Admission; on Discharge 170/70 mm. Hg.
Heart: Apex Beat 5th left space, internal to Midclavicular line. Heart sounds:- Feeble on admission. Later - 2nd aortic accentuated.
Respiratory System:- 20 per min. Lungs - Normal. No basal crepitations.
Investigations:- E.C.G. Anterior Infarct, when patient was fit, the XRay of heart taken, which revealed Calcification and Unfolding of the aorta.
Fundal Exam.:- Grade I.; Urine Exam. Volume 1200; sp.gr. 1015; Albumin - Nil. Sugar - Trace of sugar present. Later volume 1200.
Diagnosis:- Atheroma of the Aorta.
Treatment:- (1) Complete physical and mental rest; (2) Morphia initially ¼ gr. s.c.i. Rest in bed till Infarct healed; (3) Light Diabetic diet of

1000 calories.
Result:- The patient discharged symptom-free. The Pulse rate 68 on discharge; The B.P. 170/70 mm. Hg. on Discharge.

Case 72:- Male, Retired, Married, wt. 9 st. 4 lbs.
Complaint:- Mental Confusion for 2 days.
History:- The patient was perfectly fit until two days ago. He noticed the weakness in his left arm, and became mentally confused. Throbbing Frontal Headache for about ½ hour prior to the onset of his confusion.
Previous Illness:- Left-sided Sciatica 15 years ago.
Habit:- Teetotller. Family History - Nil to note.
General exam.:- An elderly man, mentally confused, although fully conscious.
Cardiovascular System:- Pulse rate 80 per min., regular in time and force, volume good, vessel wall thickened and palpable. On discharge 80.
B.P. 190/90 mg. Hg. on admission; on discharge 170/65 mm. Hg. Apex Beat: 6th left space, outside the mid-clavicular line.
Auscultation:- Soft Mitral systolic murmur and 2nd sound at aortic area accentuated.
Respiratory System:- 22 per min. No Basal Crepitations.
C.N. System:- Left sided paresis of arm, leg and face. Reflexes are exaggerated on the left side with left Extensor Plantor responses. No sensory disturbance on the left side.
Investigations:- X-Ray of Heart:- Left Ventricular Hypertrophy and calcification and Unfolding aorta; E.C.G.:- Left axis deviation. Fundal Exm.: Grade I. Urine Exam.: Incontinence at first; Later putput 1800 c.c. sp.gr. 1018;
Diagnosis:- Cerebral Thrombosis of right middle cerebral artery involving the Internal Capsule.
Treatment:- Rest and Physiotherapy.
Result:- Made very good recovery.

Case 73:- Male, 64, married, Engineer, wt. 8 st. 11 lbs.
Complaint:- Epigastric pain for 8 years and vomiting for 3 days. The patient gave typical history of Peptic ulcer. Pain relieved by alkalies and food. No history of Headache. Occasionally vomited gastric content with relief of pain. Occasional constant frontal headache after vomiting.
Previous Illness:- None till onset of his gastric trouble. Habit:- Moderate drinker but heavy smoker.
Family History:- Nothing to note.
Gener Exam.:- An elderly man who looks healthy.
Pulse Rate:- 90 per min., regular in time and force. Volume good. Arterial wall thickened and palpable and tortuous. B.P. 170/70 mm. Hg. on admission.

Apex Beat:- 5th left space, just outside Mid-clavicular line.
Auscultation:- Aortic 2nd sound accentuated,
Alimentary System:- Tongue dry and dirty. Marked splashing in the stomach with tenderness in Epigastrium. Liver and spleen - Not palpable.
Respiratory System:- 20 per min. Lungs normal.
Investigations:- X-Ray after Barium Meal: Hypo-chlorhydrics present. Stool Examination:- Benze-dine positive. X-Ray of Heart:- Calcification and Unfolding of Aorta; E.C.G. Left axis deviation.
Fundal Exm.: Stage I. Urine examination: Output 1020, sp.gr. 1020. Albumin, sugar, nil.
Co_2 Combining power 90 vols%.
Diagnosis:- Atheroma of the Aorta and Alkalosis.
Treatment:- Initially Medical for peptic ulcer, milk diet (2 hourly) with alkali, Mag. Trisilicate between feeds. Phenobarbitone gr. i. T.i.d. Increasing doses of Tinct. Belladonna 15 minims T.i.d. initially, but had no effect on stenosis.
The patient improved and was operated on for Pyloric Stenosis. At operation ulcer of the 1st part of duodenum with fibrosis and Pyloric stenosis found. The patient stood the operation well. Partial gastrectomy carried out.

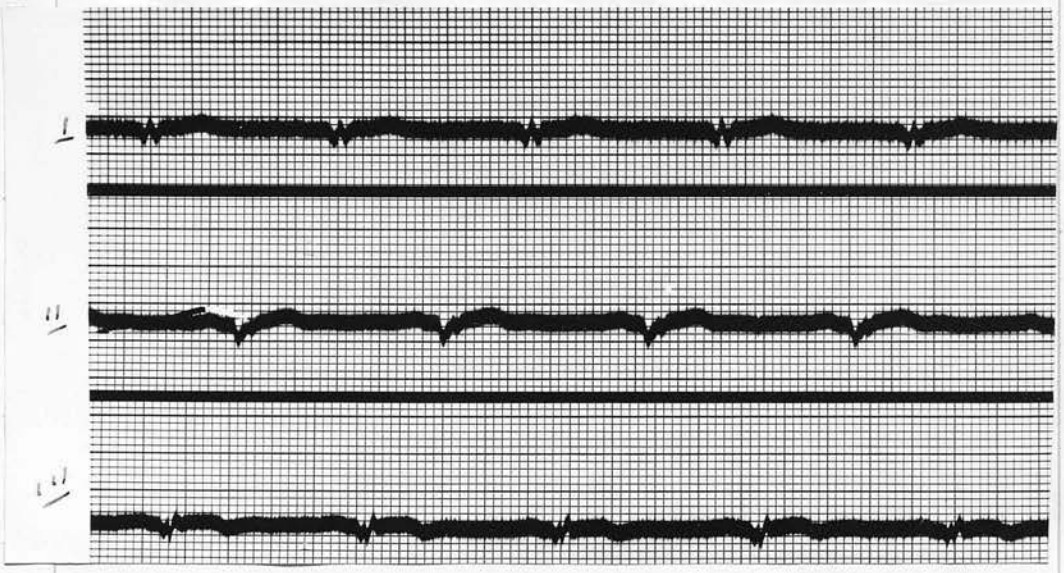
Case 74:- Female, 69, married, Housewife, wt 6 st. 5 lbs.
Complaint:- Thirst for 8 weeks. Left-sided sciatica for 1 month. Eight weeks ago, the patient began to be very thirsty and passing lot of urine. For the last month she had pain down the back of left leg. No history of headache.
Previous Illness:- Gastro-enterostomy 1911 for Peptic Ulcer.
Family History:- Husband, died 19 years ago from "Bronchitis". One son aged 40, alive and well.
General Exam.:- An elderly lady with obvious pain in her left leg.
C.V. System:- Pulse rate 82 per min. Regular in time and force. Good volume. Arterial wall thick-ened and palpable. B.P. 170/90 mm. Hg. on dis-charge;
Apex Beat: In 5th left space within mid-clavicular line. Auscultation: H. sounds normal except 2nd aortic accentuated.
Respiration system:- 18 per min. Normal.
C.N. System:- Left ankle-jerk - absent. Diminish-ed sensation to pain stimuli on outside of left foot.
Investigations:- X-Ray of Heart: Unfolding of the aorta; E.C.G. Left axis deviation; Fundal Exm.:- Stage I. Urine examination: Output 1500 c.c. sp..gr. 1030; sugar ++ ; Albumin - Nil; Acetone - Nil.
Diagnosis:- Diabetis Mellitus and left-sided Sciatica.
Treatment:- (1) Dietetic: 1800 calories diet (175 Gms. of Carbohydrate) and 10 units zinc protamine Insulin a day; (2) Aspirin grs. X. T.i.d. to re-lieve pain.

Result:- Sciatica improved once she became sugar-free.

Case 75:- Male, 64, Married, Mechanic; wt. 11 st. 13 lbs.
Complaint:- Pain in left hip for 6 months.
History:- The patient was perfectly fit till 6 months ago, when he got a 'chill'. Since then had pain in his left hip on walking. The pain in hip also occurs while in bed at night.
No History of Headache.
Previous Illness: Peptic Ulcer at the age of 48.
Habit:- Smokes pipe and Teetotaller.
Family History:- Nothing to note.
General Exam.:- A heavily-built man who apparently looks healthy. No oedema and no visual pulsation in neck.
Cardiovascular System:- Pulse rate 80 per min. regular in time and force, good in volume. Arterial wall thickened and palpable.
B.P. 160/85 mm. Hg. on admission.
Apex Beat:- 5th space just outside Midclavicular line.
Auscultation:- Aortic systolic murmur and 2nd aortic accentuated.
Respiratory System:- Respiration - 18 per minute; Lungs - Normal.
Locomotor System:- Severe pain in left hip-joint during movement, but no swelling detected in the affected side.
Investigations:- (1) X-Ray of Pelvis:- Gross osteo-arthritis of both hip-joints; (2) X-Ray of Heart: Unfolding of aorta with Calcification; (3) E.C.G.: Left axis deviation; (4) Urine Examination:- Output 1400 c.c. all throughout; sp.gr. 1015; albumin and sugar - Nil.
Diagnosis:- Bilateral Osteo-Arthritis of hip-joints a nd Arterio-sclerosis and Atheroma of the aorta.
Treatment:- (1) Diet 1000 calories (2) Heat; (3) Physiotherapy; (4) Aspirin grs. x. T.i.d.
Result:- Improved and pain disappeared.

Case 76:- Male, 84, Retired Engineer, wt. 7 st. 13 lbs.
Complaint:- Collapsed.
History: The patient was found in his own home in a collapsed state by his neighbour; the patient was a poor witness and was unable to give a good history, but he was obviously suffering from Paralysis Agitans.
No Headache.
General examination:- The patient presents a typical picture of Parkinsonism.
Cardio-vascular System:- Pulse Rate 100 on admission; Regular in time and force. Good in volume and arterial wall palpable and thickened and calcified.

CASE N° 77



B.P. 190/80 mm. Hg. on admission; Apex Beat: 6th left interspace ½ inch outside the mid-clavicular line.
Auscultation:- Mitral Systolic Murmur; Aortic Systolic Murmur; 2nd Aortic markedly accentuated. Respiratory System:- 24 per min. Generalised Ronchi in both lungs.
C.N. System:- Rolling of hands; Cog-wheel spacticity; all reflexes are present.
Investigations:- (1) X-Ray of Heart: Calcification of Aorta; Unfolding of aorta; Left Ventricle enlarged; (2) E.C.G. Left axis deviation; (3) Blood Urea N_2 20 mgm%.; (4) Fundal Exm.: Grade I. (5) Urine exm.: Output 1800 c.c. sp.gr. 1012; albumin - Trace; Casts - Nil.
Diagnosis:- Cerebral Arteriosclerosis with Paralysis Agitans.
Treatment:- Tinct. stramonium m x. T.i.d. and was discharged to an Old Persons' Home without any dramatic improvement.

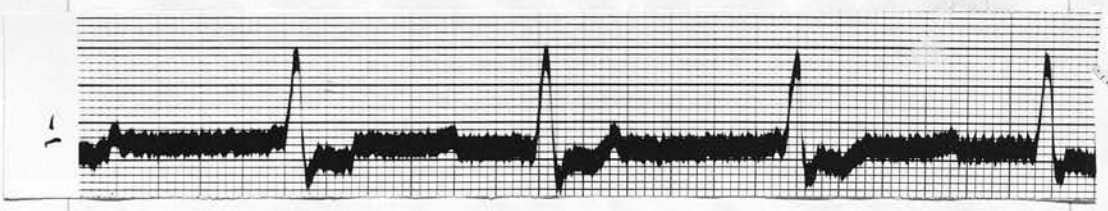
Case 77:-Female, 69, widow; Household; wt. 8 st.
Complaint:- Headache for 4 days; Insomnia for 6 months.
History:- The patient's history was obtained from her daughter as the patient was a little confused.
For the last six months the patient has been wandering about the house at night and sleeping during the day. For the last 4 days complained of throbbing frontal headache worse on sitting up and by sudden movement. Headache relieved by rest in bed. The attacks lasted for a few minutes.
Habit:- Nil.
Previous Illness:- Operated for gall stones 10 years ago.
Family History:- Husband died /of /Coronary Thrombosis 5 years ago; children - 4 - alive and well.
General Examination:- An elderly lady somewhat confused. Pulse rate:- 90 per min. regular in time and force, volume good, vessel wall thickened, calcified and palpable. B.P. 190/80 mm.Hg. on admission. Apex Beat:- 5th left space outside Mid-clavicular line.
Ausculation:- 2nd aortic accentuated.
C.N. System - Normal.
Respiratory System:- Respiration: 20 per minute. No basal crepitation.
Investigations:- (1) X-Ray of Heart:- Unfolding of the aorta; calcification of the aorta; Left Ventricle enlargement; (2) E.C.G.: Showing Low Voltage; (3) Fundal Exam.:- Stage I.; (4) C.S.Fluid: Pressure 120 millimeter of H_2O (Normal). Protein 20 mgm%.; Chloride 700 mgm%.; Sugar 80 mgm%.; R.B.C. None. W.B.C. 2 per c. mm. W.R. - Negative.
Urine examination: Output 1600 c.c.;on discharge the same; sp.gr. 1018, albumin & sugar - Nil.

Diagnosis:- Cerebral Arterio-Sclerosis and Atheroma of the Aorta and Aortic cusps.
Treatment:- (1) Bed rest; Headache responded well.
Chloral grs. xx. at night.
Tinct. opii m xx. to secure sleep.
Result:- Headache relieved and the patient dischanged symptom-free.

Case 78:- female, 85.
Present Complaint:- Difficulty in walking for 8 years. Headache and buzzing noise in head for 16 years; trouble with her eyesight for 1 year.
History:-Sixteen years ago, the patient began to suffer from Insomnia which was largely due to Throbbing Frontal Headache and buzzing in the head when she lay down at night in bed. Her doctor used to give medicine to relieve it.
For the last eight years she has been unsteady on walking. For the last year her eyesight began to trouble her.
Previous Illness:- Pneumonia 40 years ago.
Family History:- Husband died 10 years ago of heart attack; Children - 3 - all alive and well.
General Exm.: An elderly lady looks apparently healthy.
Cardiovascular System:- Pulse Rate 100 per min., irregular in time and force. Vessel wall thickened and palpable. B.P. 210/90 mm. Hg. on admission.
Heart:- Apex Beat:- 6th left Interspace outside midclavicular line.
Auscultation:- Systolic murmur and 2nd aortic accentuated.
Respiratory System:- 20 per min. Lungs - Normal.
C.N. System:- Signs of left Cerebellar Lesion, viz.,Nystagmus on looking to the left. Past-pointing to the left. Intention tremors on the left side and an Ataxia.
Investigations:- (1) X-Ray of Heart:- Enlarged left ventricle; Calcified and Unfolding of Aorta; (2) E.C.G.: Left axis deviation; (3) Blood Urea N_2:- 22 mgm%.- Fundal exm.: Stage I.; (5) Urine Examination: Output 1500 c.c. sp.gr. 1010. Albumin - Trace. Sugar - Nil; (6) C.S. Fluid - Normal. Pressure 130 H_2O. Protein 30 mgm%; Sugar 85 mgm%. Chloride 720 mgm%. Cells - None.
W.R. - Negative.
Diagnosis:- Generalised Arteriosclerosis with Cerebellar Arteriosclerosis, and Atheroma of the aorta and aortic cusps.

Treatment:- (1) Bed rest; (2) Chloral Hydrate grs. xx. at night; (3) Phenobarbitone gr. ½ T.i.d.
Kept in ward for a week and discharged to her home without any improvement.
Result:- Headache - not relieved, and remains just the same.

CASE N° 80



Cases of Complete Heart Block.

Case 79:- Man, 70, Distillery Labourer, married, wt. 9 st. 5¾ lbs.
Complaint:- (1) Fainting attack once; (2) No Headache.
Previous Illness:- Had several attacks of Influenza; No history of Giddiness previously or of Rheumatic Fever. Habit:- Pipe-smoker.
Family History:- Father died of cancer of the tongue; Mother died of old age; wife and children all alive and well.
On Examination: A pleasant-co-operative and unusually bright individual.
Cardiovascular System:- Pulse Rate: 31 per min, regular and radial, palpable from wrist to elbow.
Blood Pressure :- 190/90 mm. Hg. on admission.
Heart: Apex Beat:- 5th left intercostal space one inch outside the mid-clavicular line.
Auscultation:- Variation of 1st sound resembling "cannon"ball sound.
Investigations:- (1) X-Ray of Heart - Enlarged left ventricle; (2) E.C.G.:- Left Bundle-Branch block and complete heart-block; (3) Wassermann Reaction - Negative; (4) Fundal exam.:- Grade I., Arteriosclerotic; (5) Urine:- Normal.
Diagnosis:- Left bundle branch block with complete heart-block due to atheromatous coronary arteries.
Treatment:- (1) Complete physical and mental rest; (2) Ephedrine Hydrochloride gr ½ T.i.d. orally for 3 weeks.
Result:- Discharged unrelieved. B.P. remains the same 190/90 mm. Hg. Syncope - none while in Hospital. Pulse rate - same.
Discharged unrelieved.

Case 80:- Female, 35, married, wt. 8 st. 5 lbs.
Occupation:- Housewife.
Complaint:- (1) Attacks of faintings for the last 3 years; (2) Throbbing frontal headache on exertion, relieved by rest. Duration - 3 months.
Previous Illness:- (1) Diphtheria at the age of 8 years. After the attack of diphtheria, she complained of shortness of breath on exertion and has always felt that there was something wrong with her. No history of Rheumatic fever. Menstruation regular.
Habit - Nil.
Family History:- Father died of accident; Mother aged 63, alive and well. No children. Brother aged 30 alive and well; sister aged 28 alive and well.

~~Case 81.~~

On examination: A thin, agitated, nervous and apprehensive.
Cardiovascular System:- Pulse rate - 48 per min., regular, vessel wall not palpable. B.P. 160/70 mm. Hg. on admission. Distended neck vein.
Heart:- Apex Beat:- 5th left Interspace within mid-clavicular line.
Auscultation:- Changing intensity in sounds.
Investigations:- (1) X-Ray of Heart - Nil to note; (2) E.C.G.:- Complete heart-block; (3) W.R. - Negative; (4) Fundus Oculi - Normal; (5) Urine :- Normal;
Diagnosis:- Complete heart-block, presumably due to diphtheria?
Treatment:- Complete bed rest and mental rest; (2) Ephedrine Hydrochloride gr. ½ T.i.d. for 3 wks.
Result:- Ephedrine checked the fainting attacks, but no effect on pulse rate. Headache relieved. B.P. 140/70 on discharge. Pulse Rate - 48 per min. Regular on discharge.

Case 81:- Male, 62, Coal-miner. married, wt. 9 st.5 lbs.
Complaint:- (1) Attacks of fainting for the last 11 months - nearly 40 attacks a day.
History:- (One year ago, complained of precordial pain radiating to left arm and which was treated as neuritis - probably ischaemic pain due to coronary disease.
Previous Illness:- Gastritis. No history of Headache.
Habit:- Was heavy drinker, but became teetotaller after gastric trouble.
Family History:- Father died at 73 of cerebral haemorrhage; Mother died at 71 of heart-attack. Daughter aged 32, alive and well.
On examination: An elderly man of good physique.
Cardiovascular system:- Pulse rate - 38 per min. regular, vessel wall palpable but not tortuous. B.P. 220/90 mm. Hg. on admission. Visible pulsation in neck. Apex Beat:- 5th left interspace 2 inch outside the Mi-clavicular line.
Auscultation:- Changing intensity in sounds. Aortic systolic murmur conducted into the vessels of the neck.
Investigations:- (1) X-Ray of Heart: Nil to note ; (2) E.C.G.: Complete heart-block and left bundle branch block; W.R. - Negative. (4) Fundi: Grade I.
Urine:- Nil to note.
Diagnosis:- Complete heart-block and left bundle-branch block due to atherosclerosis of coronary arteries.
Treatment:- (1) Complete mental and physical rest; (2) Ephedrine Hydrochloride gr. ½ T.i.d.

Result:- (1) Syncopal attacks checked by Ephedrine; (2) Blood pressure 210/90 mm. Hg. on discharge; (3) No increase of Pulse rate by Ephdrine.

Case 82:-

Female, 74; Married, wt. 9 st. 12 lbs.
Occupation: Housewife.
Complaint:- Breathlessness, worse on exertion and fainting attacks started three months ago, while at rest, without any obvious cause to account for them. Breathlessness on exertion for the last three years, but no history of Pre-cordial pain.
Family History:- Husband aged 72, alive and well;
Brother died at 62 years, cancer of throat;
Brother died at 56 years - Cancer of Lung;
Brother died at 73 years - Heart Disease.
Sister died at 34 years - Pulmonary Tuberculosis.
Previous Illness:- Nil, Menopause at usual age.
No History of Headache.
Habit - Nil.
On examination:- Lady appeared younger than her age, with bright complexion.
Cardiovascular System:- Pulse rate - 36 per min, regular, vessel wall palpable. B.P. 210/85 mm. Hg. on admission. Apex Beat:- 5th left Intercostal space just on Mid-clavicular line.
Auscultation:- Changing intensity in sounds. A harsh aortic systolic murmur propagated into the neck.
Investigations:- (1) X-Ray of Heart:- Calcification of aorta and left ventricular enlargement; (2) E.C.G.: Complete heart-block and left axis deviation; (3) Fundal exam.:- Grade I.; (4) Urine: Normal. (5) W.R. Negative.
Diagnosis:- Complete Heart-block and left bundle-branch block due to Atherosclerosis of coronary artery.
Treatment:- (1) Physical and mental rest;
(2) Ephedrine Hydrochloride gr. ½ T.i.d. while under treatment;
Result:- Fainting attacks relieved. B.P. 210/85 mm. Hg. on discharge; Pulse rate 36 per min. regular.
